********** FILE 'USPAT' ENTERED AT 15:40:17 ON 05 OCT 94

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 ********************************** *          WELCOME TO TH
E          * *     U.S. PATENT TEXT FILE       * **********
**********
=> s (cancer or tumor)(p)(irradiat?)
      12446 CANCER
       9929 TUMOR
      56323 IRRADIAT?
L1      840 (CANCER OR TUMOR)(P)(IRRADIAT?)

=> s l1 and melanoma
       1857 MELANOMA
L2       95 L1 AND MELANOMA

=> s l2 and cyclophosphamide
        877 CYCLOPHOSPHAMIDE
L3       13 L2 AND CYCLOPHOSPHAMIDE

=> d l3 1-13
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1. 5,290,551, Mar. 1, 1994, Treatment of melanoma with a vaccine comprising irradiated autologous melanoma tumor cells conjugated to a hapten; David Berd, 424/193.1, 85.2, 277.1 [IMAGE AVAILABLE]

2. 5,239,062, Aug. 24, 1993, Blocked lectins, methods and affinity support for making same using affinity ligands, and method of killing selected cell populations having reduced nonselective cytotoxicity; Walter A. Blattler, et al., 530/396; 424/179.1, 183.1; 530/370, 391.7, 402, 408, 409 [IMAGE AVAILABLE]

3. 5,192,553, Mar. 9, 1993, Isolation and preservation of fetal and neonatal hematopoietic stem and progenitor cells of the blood and methods of therapeutic use; Edward A. Boyse, et al., 424/529; 435/2, 172.1, 172.3, 240.2, 240.26 [IMAGE AVAILABLE]

4. 5,167,956, Dec. 1, 1992, Immunotoxin with in-vivo T cell suppressant activity; David M. Neville, Jr., et al., 424/183.1, 173.1; 530/391.7, 391.9 [IMAGE AVAILABLE]

5. 5,164,196, Nov. 17, 1992, Crotoxin complex as cytotoxic agent; Guillermo J. H. Plata, et al., 424/542; 514/2, 21; 530/856 [IMAGE AVAILABLE]

6. 5,126,132, Jun. 30, 1992, Tumor infiltrating lymphocytes as a treatment modality for human cancer; Steven A. Rosenberg, 424/85.2, 93.71, 534 [IMAGE AVAILABLE]

7. 5,023,172, Jun. 11, 1991, Predictive assay for tumor control; Bozidar Djordjevic, 435/29, 30, 32, 39, 172.2, 173.1, 240.2, 240.22, 242, 243 [IMAGE AVAILABLE]

8. 5,004,681, Apr. 2, 1991, Preservation of fetal and neonatal hematopoietic stem and progenitor cells of the blood; Edward A. Boyse, et al., 435/2; 424/529 [IMAGE AVAILABLE]

9. 4,985,241, Jan. 15, 1991, Therapeutic combination of free-radical scavenger and tumor necrosis factor; Robert Zimmerman, et al., 424/85.1, 85.2; 514/2, 8, 885 [IMAGE AVAILABLE]

10. 4,889,525, Dec. 26, 1989, Sensitization of hypoxic tumor cells and control of growth thereof; John M. Yuhas, et al., 600/1; 514/644, 756; 600/2 [IMAGE AVAILABLE]

11. 4,742,050, May 3, 1988, Sensitization of hypoxic tumor cells and control of growth thereof; John M. Yuhas, deceased, et al., 514/34 [IMAGE AVAILABLE]

12. 4,614,811, Sep. 30, 1986, Novel organoplatinum(II) complexes and method for the preparation thereof; Ottavio Gandolfi, 556/137; 987/11 [IMAGE AVAILABLE]

13. 4,481,137, Nov. 6, 1984, Glycoproteins and processes for their production; Haruo Ohnishi, et al., 530/395; 424/85.1; 435/70.3, 70.4; 514/8, 908; 530/322, 351, 806, 809, 828, 829 [IMAGE AVAILABLE]
=> d l3 6 cit ab

6. 5,126,132, Jun. 30, 1992, Tumor infiltrating lymphocytes as a treatment modality for human cancer; Steven A. Rosenberg, 424/85.2, 93.71, 534 [IMAGE AVAILABLE]

US PAT NO: 5,126,132 [IMAGE AVAILABLE] L3: 6 of 13
ABSTRACT:
A new immunotherapeutic method of treating cancer with a combination of tumor infiltrating lymphocytes and IL-2 has been described.
=> d l3 6 kwic

US PAT NO: 5,126,132 [IMAGE AVAILABLE] L3: 6 of 13
DRAWING DESC:

DRWD(6)

FIG. 4: Photographs of a 21-year old male who had a large recurrent growth of melanoma in his left neck following a radical neck dissection. FIG. 4a shows the neck mass prior to treatment and FIG.. . .

DRAWING DESC:

DRWD(8)

FIG. 6: Photographs of the chest well of a 38-year old woman who had multiple cutaneous metastases following resection of melanoma . FIG. 6a shows a large cutaneous metastases on the right chest wall as well as a large growing metastatic deposit. . .

DETDESC:

DETD(6)

Patients . . . cytokines. In 20 patients reported in the New England Journal of Medicine 319:1676-1680, 1988, objective reference regression of advanced cancer (melanoma) was seen in 11 patients. Of 15 patients that had previously not been treated with immunotherapy, nine responded (60%). Of. . .

DETDESC:

DETD(10)

All the patients had a diagnosis of metastatic malignant melanoma , which could be evaluated by physical or radiographic examination. Of the 20 patients in this study, 18 had undergone surgical. . .
DETDESC:

DETD(19)

Tumor . . . to techniques described herein supra. When the TIL were ready for infusion, patients first

received a single intravenous dose of cyclophosphamide (25 mg per kilogram of body weight) and 36 hours later the first intravenous infusion of TIL in an intensive. . .
DETDESC:

DETD(23)

Studies in murine tumor models had indicated that successful therapy with TIL depended on prior administration of cyclophosphamide , although this is not necessarily required in humans (Rosenberg et al, Science, 1986; 223:1318-21). Thus, to determine the degree of tolerance and response to cyclophosphamide plus interleukin-2 without administration of TIL, these clinical studies were begun by treating a series of 13 patients with metastatic melanoma with various doses of cyclophosphamide (4 patients with 50 mg per kilogram, 6 with 25 mg per kilogram, and 3 with 10 mg per kilogram). . .

DETDESC:

DETD(24)

The characteristics of the 20 patients with metastatic melanoma treated with cyclophosphamide , TIL, and interleukin-2 and the characteristics of their treatment and response are shown in Table 1. The number of TIL. . . bone, and subcutanneous tissue. Two of these responding patients (Patients 8 and 9) received a second course of therapy with cyclophosphamide , TIL, and interleukin-2, and four (Patients 3 through 6) received a second course of interleukin-2 alone approximately two months after the first course of cyclophosphamide , TIL, and interleukin-2. All these patients, however, had objective responses after the first course of treatment. The duration of the. . .
DETDESC:

DETD(27)

Studies of the adoptive transfer of TIL in murine tumor models have shown that these cells are 50 to 100 times more effective than LAK cells in mediating tumor regression (Rosenberg et al, Science, 1986, 223:1318-21; Spiess et al, JNCI, 1987; 79:1067-75). In contrast to LAK cells, TIL obtained from mice and patients are predominantly T lymphocytes, and those from patients are often capable of lysing autologous melanoma in a fashion that is highly specific and restricted by the major histocompatibility complex (Muul et al, J Immunol, 1987, 138:989-95; Itoh et al, Cancer Res, 1986, 46:3011-7; Kurnick et al, Clin Immunol Immunopathol, 1986, 38:367-80; Rabinowich et al, Cancer Res, 1987, 47:173-7; Miescher et al, J Immunol, 1987, 138:4004-11; Topalian et al, J Immunol Methods, 1987, 102:127-41; Belldegrun et al, Cancer Res, 1988, 48:206-14). As with other forms of experimental adoptive therapy with T cells, immunosuppression of the tumor -bearing host with either cyclophosphamide or total-body irradiation is required for treatment to be successful (Berendt et al, J Exp Med, 1980, 151:69-80; Shu et al, J Immunol,. . . et al, J Exp Med, 1982, 156:385-97). This pretreatment is thought to eliminate suppressor cells or to facilitate lymphocyte "homing."

Cyclophosphamide administration or total-body irradiation does not affect treatment with LAK cells in murine models.

DETDESC:

DETD(28)

Therefore, 13 patients were first treated with the combined administration of cyclophosphamide and interleukin-2, and observed only two objective responses (15 percent), in accord with the response expected with the use of interleukin-2 alone. However, the addition of TIL to the combination of cyclophosphamide and interleukin-2 resulted in responses in 9 of 15 patients (60 percent) who had not previously been treated with interleukin-2. . . a different preparation of recombinant interleukin-2). It thus appears that treatment with TIL increased response rates among patients with metastatic melanoma , as compared with therapy with LAK

cells and interleukin-2, cyclophosphamide and interleukin-2, or interleukin-2 alone. The results reported here reflect primarily the results of a single cycle of treatment with. . .
DETDESC:

DETD(30)

Extensive . . . the TIL populations that will mediate cancer regression in vivo. Moreover, a study of TIL traffic in six patients with melanoma who each received a small aliquot of indium-111-labeled TIL revealed subs antial homing of TIL to cancer deposits [Fisher et. . .
CLAIMS:

CLMS(4)

4. The method of claim 2 wherein an effective amount of
cyclophosphamide is administered to patient prior to TIL-IL2 infusion.

CLAIMS:

CLMS(5)

5. The method of claim 4 wherein the amount of cyclophosphamide ranges from about 10 to 50 mg per kilogram body weight.

CLAIMS:

CLMS(6)

6. The method of claim 1 wherein said cancer is melanoma , lung, liver, cutaneous or subcutaneous metastastases.

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(FILE 'USPAT' ENTERED AT 15:40:17 ON 05 OCT 94)
L1 840 S (CANCER OR TUMOR)(P)(IRRADIAT?)
L2 95 S L1 AND MELANOMA
L3 13 S L2 AND CYCLOPHOSPHAMIDE

=> s l1 and hapten
1759 HAPTEN
L4 17 L1 AND HAPTEN

=> d l4 1-17

1. 5,342,604, Aug. 30, 1994, Complexes possessing ortho ligating functionality; David A. Wilson, et al., 424/1.65; 534/10 [IMAGE AVAILABLE]

2. 5,302,389, Apr. 12, 1994, Method for treating UV-induced suppression of contact hypersensitivity by administration of T4 endonuclease; Margaret L. Kripke, et al., 424/94.6, 94.3, 450 [IMAGE AVAILABLE]
3. 5,290,551, Mar. 1, 1994, Treatment of melanoma with a vaccine comprising irradiated autologous melanoma tumor cells conjugated to a hapten ; David Berd, 424/193.1, 85.2, 277.1 [IMAGE AVAILABLE]
4. 5,262,177, Nov. 16, 1993, Recombinant viruses encoding the human melanoma-associated antigen; Joseph P. Brown, et al., 435/235.1; 424/185.1, 199.1, 232.1; 435/69.3, 172.3, 240.2, 252.3, 252.33, 320.1; 530/350; 536/23.5; 935/9, 32, 41, 57, 65, 70, 73 [IMAGE AVAILABLE]

5. 5,256,395, Oct. 26, 1993, Affinity enhancement immunological reagents for in vivo detection and killing of specific target cells; Jacques Barbet, et al., 424/9, 1.57, 136.1, 193.1, 194.1 [IMAGE AVAILABLE]
6. 5,210,072, May 11, 1993, Muramyl dipeptide derivatives; Louis Chedid, et al., 514/8, 18; 530/322, 331; 536/53 [IMAGE AVAILABLE]

7. 5,141,742, Aug. 25, 1992, Vaccines against melanoma; Joseph P. Brown, et al., 424/186.1, 277.1; 435/69.3, 70.1, 71.1, 71.2; 530/350, 395; 536/23.5 [IMAGE AVAILABLE]

8. 5,095,095, Mar. 10, 1992, Immunosuppressant factor protein capable of inhibiting T-cell mechanisms; Adriano Fontana, 530/350, 351, 827, 839 [IMAGE AVAILABLE]

9. 5,057,598, Oct. 15, 1991, Monoclonal antibodies reactive with endotoxin core; Matthew Pollack, et al., 424/150.1, 803; 435/70.21, 172.2, 172.3, 240.2, 240.26, 240.27; 530/388.4, 806, 808, 825 [IMAGE AVAILABLE]

10. 5,045,320, Sep. 3, 1991, Large multivalent immunogen; Matthew F. Mescher, 424/450, 9, 85.1, 85.2, 85.4, 184.1, 193.1, 277.1, 812; 436/527 [IMAGE AVAILABLE]

11. 4,925,922, May 15, 1990, Potentiation of cytotoxic conjugates; Vera S. Byers, et al., 424/182.1, 156.1, 183.1; 435/172.2, 240.27; 530/388.85, 391.7, 806, 808, 828 [IMAGE AVAILABLE]

12. 4,918,164, Apr. 17, 1990, Tumor immunotherapy using anti-idiotypic antibodies; Ingegerd Hellstrom, et al., 530/387.2; 424/131.1, 156.1; 435/172.2, 240.27; 530/388.85, 395, 806, 808, 828, 866 [IMAGE AVAILABLE]
13. 4,916,213, Apr. 10, 1990, Ribosomal inhibiting protein- immunoglobulin conjugates with specificity for tumor cell surface antigens, and mixtures thereof; Patrick J. Scannon, et al., 424/183.1, 156.1; 435/172.2, 240.27; 530/388.85, 391.7, 806, 808, 828 [IMAGE AVAILABLE]

14. 4,789,658, Dec. 6, 1988, Immunoprophylactic and immunotherapeutic agents; Ryota Yoshimoto, et al., 514/2; 435/69.52, 70.2, 70.3, 70.4; 514/8, 12; 530/351, 370, 371 [IMAGE AVAILABLE]

15. 4,672,106, Jun. 9, 1987, Muramylpeptide active ester derivatives; Toshiyuki Hamaoka, et al., 530/331; 536/53; 930/DIG.500 [IMAGE AVAILABLE]
16. 4,659,655, Apr. 21, 1987, Method for isolating product-producing cells; Sam Rose, 435/7.21, 7.23, 7.24, 7.25, 177, 178, 182, 803, 966; 436/512, 520, 521, 522, 535, 536, 539, 821, 824 [IMAGE AVAILABLE]
17. 4,639,420, Jan. 27, 1987, Method for the immunoanalysis of cholesterol epoxides; Carl P. Schaffner, 435/7.1, 11, 15, 961; 436/501, 543, 817, 822, 823 [IMAGE AVAILABLE]

=> d l4 5,7,12

5. 5,256,395, Oct. 26, 1993, Affinity enhancement immunological reagents for in vivo detection and killing of specific target cells; Jacques Barbet, et al., 424/9, 1.57, 136.1, 193.1, 194.1 [IMAGE AVAILABLE]
7. 5,141,742, Aug. 25, 1992, Vaccines against melanoma; Joseph P. Brown, et al., 424/186.1, 277.1; 435/69.3, 70.1, 71.1, 71.2; 530/350, 395; 536/23.5 [IMAGE AVAILABLE]

12. 4,918,164, Apr. 17, 1990, Tumor immunotherapy using anti-idiotypic antibodies; Ingegerd Hellstrom, et al., 530/387.2; 424/131.1, 156.1; 435/172.2, 240.27; 530/388.85, 395, 806, 808, 828, 866 [IMAGE AVAILABLE]
=> d l4 5,7,12 cit ab

5. 5,256,395, Oct. 26, 1993, Affinity enhancement immunological reagents for in vivo detection and killing of specific target cells; Jacques Barbet, et al., 424/9, 1.57, 136.1, 193.1, 194.1 [IMAGE AVAILABLE]
US PAT NO: 5,256,395 [IMAGE AVAILABLE] L4: 5 of 17
ABSTRACT:

Immunological reagents, consisting of a) antibody or fragment conjugates having both an anti-cell specificity and an anti- hapten specificity, and b) synthetic tracers containing at least two hapten sites and at least one site suitable to attach radioactive isotopes, paramagnetic ions, drugs or toxins, are provided. These reagents are capable of binding to target cells in a specific way, and the tracer localizes preferentially on the membrane of antigen-bearing cells, even in the presence of excess antibody conjugate (affinity enhancement). These reagents are usefully employed, either in vitro or in vivo, to detect tumors, metastases, or other tissue injuries, when the synthetic tracer carries radioactive or paramagnetic compounds, and to kill target cells when the synthetic tracer carries radioactive compounds or drugs or toxins.

7. 5,141,742, Aug. 25, 1992, Vaccines against melanoma; Joseph P. Brown, et al., 424/186.1, 277.1; 435/69.3, 70.1, 71.1, 71.2; 530/350, 395; 536/23.5 [IMAGE AVAILABLE]

US PAT NO: 5,141,742 [IMAGE AVAILABLE] L4: 7 of 17
ABSTRACT:
Peptides or proteins related to a melanoma associated antigen are described. These are produced in large quantities via recombinant DNA techniques and/or by chemical synthetic methods. The peptides or proteins can be used as immunogens in vaccine formulations which can induce an immune response that selectively destroys melanoma cells in a vaccinated individual. Where the peptides or proteins are expressed by a recombinant virus, inactivated or live virus vaccine formulations may be prepared.
12. 4,918,164, Apr. 17, 1990, Tumor immunotherapy using anti-idiotypic antibodies; Ingegerd Hellstrom, et al., 530/387.2; 424/131.1, 156.1; 435/172.2, 240.27; 530/388.85, 395, 806, 808, 828, 866 [IMAGE AVAILABLE]
US PAT NO: 4,918,164 [IMAGE AVAILABLE] L4: 12 of 17
ABSTRACT:
The present invention relates to methods which utilize anti-idiotypic antibodies, or fragments thereof, for tumor immunotherapy or immunoprophylaxis. Monoclonal anti-idiotypic antibodies which recognize an idiotype present on a second antibody or on a T lymphocyte or on an immune suppressor factor which is directed against a defined tumor antigen, can be used for immunization against a tumor, for immune anti-tumor activation or inhibition of suppression, or for in vitro activation of lymphocytes to be used in adoptive immunotherapy. The anti-idiotypic antibodies, or fragments thereof, can also be used to monitor anti-antibody induction in patients undergoing passive immunization to a tumor antigen by administration of anti-tumor antibody. In another embodiment, administration of T lymphocytes which express an idiotype directed against a defined tumor antigen can be used to transfer delayed-type hypersensitivity to the tumor. In another method of the invention, the induction of anti-idiotypic antibodies in vivo by administration of anti-tumor antibody or immune cells or factors exhibiting an anti-tumor idiotype can be therapeutically valuable.
=> s irradiat?(p)tumor(p)hapten
56323 IRRADIAT?
9929 TUMOR
1759 HAPTEN
L5 3 IRRADIAT?(P)TUMOR(P)HAPTEN

=> d l5 1-3

1. 5,290,551, Mar. 1, 1994, Treatment of melanoma with a vaccine comprising irradiated autologous melanoma tumor cells conjugated to a hapten ; David Berd, 424/193.1, 85.2, 277.1 [IMAGE AVAILABLE]
2. 5,095,095, Mar. 10, 1992, Immunosuppressant factor protein capable of inhibiting T-cell mechanisms; Adriano Fontana, 530/350, 351, 827, 839 [IMAGE AVAILABLE]

3. 4,672,106, Jun. 9, 1987, Muramylpeptide active ester derivatives; Toshiyuki Hamaoka, et al., 530/331; 536/53; 930/DIG.500 [IMAGE AVAILABLE]
=> d l5 2,3, kwic

US PAT NO: 5,095,095 [IMAGE AVAILABLE] L5: 2 of 3

DETDESC:

DETD(5)

Analogous . . . effect of the IL 1-like helper factor, however, is overriden by the presently described inhibitory factor also elaborated by the tumor cells. This factor inhibits the proliferative response of T cells to Con A 3 or to both PHA and IL 1/IL 2 standards. The factor also blocks the proliferation of an H-2-restricted, hapten -specific T cell line that normally grows in the presence of IL 2 and haptenated irradiated spleen cells. The glioblastoma-derived suppressor factor has no effect on IL 2 production by Con A-stimulated spleen cells and does. . .

US PAT NO: 4,672,106 [IMAGE AVAILABLE] L5: 3 of 3
DETDESC:

DETD(138)

For the purpose of inducing helper T cells reactive to the MDP-related hapten in mouse spleen cells, a mouse having been irradiated with 150 R X-rays was twice immunized by subcutaneously injecting 1 mg of BCG. The immunized mouse further received 5 intraperitoneal injections of the aforesaid MDP-Lys(L2)-modified syngeneic X5563 tumor cells (1.times.10.sup.7) treated with mitomycin. The immunization with the MDP-Lys(L2)-modified syngeneic tumor cells aimed at enhanced generation of T.sub.E cells reactive to a tumor associated antigen in the mouse spleen cells. Spleen cells (2.times.10.sup.7) were prepared from the treated mouse and used as a. . . controls, spleen cells from a mouse which had been treated only with 5 intraperitoneal injections of the MDP-Lys(L2)-modified syngeneic X5563 tumor cells treated with mitomycin (Control Group 1) and spleen cells from a mouse which had received no treatment at all. . .

=> logoff hold
SESSION WILL BE HELD FOR 30 MINUTES
U.S. Patent & Trademark Office SESSION SUSPENDED AT 15:47:23 ON 05 OCT 94

File 155:MEDLINE(R) 1966-1994/Nov W4
(c) format only 1994 Dialog Info.Svcs.
File 5:BIOSIS PREVIEWS(R) 1969-1994/OCT W3
(c) 1994 BIOSIS
File 73:EMBASE 1974-1994/ISS 39
(c) 1994 Elsevier Science B.V.
*File 73: Truncate EMTREE codes(e.g. DC=C1.120?) for complete retrieval. See HELP NEWS 73 for explode feature.

Set Items Description
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?s irradiat? and (tumor or cancer)

Processing
Processing
Processing
Processing
235746 IRRADIAT?
756746 TUMOR
1012282 CANCER
S1 59781 IRRADIAT? AND (TUMOR OR CANCER)
?s s1 and vaccine?

59781 S1
188293 VACCINE?
S2 1315 S1 AND VACCINE?
?s s2 and hapten?

1315 S2
21250 HAPTEN?
S3 9 S2 AND HAPTEN?
?rd

...completed examining records
S4 7 RD (unique items)
?t s4/6/1-7

4/6/1 (Item 1 from file: 155)
06505871 88150871
The augmentation of ***tumor***-specific immunity using ***haptenic*** muramyl dipeptide (MDP) derivatives. III. Eradication of disseminated murine chronic leukemia cells by utilizing MDP ***hapten***-reactive helper T-cell activity.

4/6/2 (Item 2 from file: 155)
05358790 84282790
Tumor bearer T cells suppress Bacillus Calmette-Guerin-potentiated antitumor responses. III. Identification of an auxiliary efferent suppressor-T-cell population.

4/6/3 (Item 1 from file: 73)
6386155 EMBASE No: 87122814
Studies on the recovery from tolerance to ***tumor*** antigens. II. Accelerated recovery of

tumor-specific effector T cells in tolerant mice by applying T-T cell interaction mechanism

4/6/4 (Item 2 from file: 73)
6160477 EMBASE No: 86155537
Immunological suppression in mice treated with hematoporphyrin derivative photoradiation

4/6/5 (Item 3 from file: 73)
5594518 EMBASE No: 84090184
Studies in the enhancement of tumour immunity by coupling strong antigens to tumour cells ('heterogenization of tumours'). Helper T cell clones against PPD help other T cells mount anti-tumour responses to PPD-coupled tumour cells

4/6/6 (Item 4 from file: 73)
5240184 EMBASE No: 82245919
Autologous x-***irradiated*** tumour cells and percutaneous BCG in operable lung ***cancer***

4/6/7 (Item 5 from file: 73)
5082909 EMBASE No: 82085954
Tumor bearer T cells suppress BCG-potentiated antitumor responses. II. Characteristics of the efferent phase suppressor
?t s4/7/1-7

4/7/1 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

06505871 88150871
The augmentation of ***tumor***-specific immunity using ***haptenic*** muramyl dipeptide (MDP) derivatives. III. Eradication of disseminated murine chronic leukemia cells by utilizing MDP ***hapten***-reactive helper T-cell activity.
Shima J; Yoshioka T; Nakajima H; Fujiwara H; Hamaoka T
Department of Oncogenesis, Osaka University Medical School, Japan. Cancer Immunol Immunother (GERMANY, WEST) 1988, 26 (1) p43-7, ISSN 0340-7004 Journal Code: CN3
Languages: ENGLISH
Document type: JOURNAL ARTICLE
A previous paper has demonstrated that enhanced ***tumor***-specific immunity could be induced by priming mice with Bacillus Calmette Guerin (BCG) and subsequently immunizing them with syngeneic ***tumor*** cells modified with BCG-cross-reactive muramyl dipeptide (MDP) ***hapten***. The present study establishes a ***tumor***-specific immunotherapy protocol for a murine chronic leukemia based on the above T-T cell collaboration between antitumor effector T cells and anti-MDP ***hapten*** helper T cells induced by BCG priming. BALB/c mice which had been primed to BCG were injected intravenously (i.v.) with viable, syngeneic BCL1 leukemia cells. One week later, these mice were immunized intraperitoneally (i.p.) with unmodified or MDP ***hapten***-modified, 10,000 R X-***irradiated*** BCL1 cells, followed by 4 booster immunizations at 5-day intervals. The administration of unmodified BCL1 ***tumor*** cells into BCG-primed mice failed to prevent them from ***tumor*** death due to the persistent growth of preinjected BCL1 cells. In contrast, the immunization of BCG-primed, BCL1 leukemia-cell-bearing mice with MDP-modified BCL1 cells resulted in a high growth inhibition of leukemia cells and protection of these mice from death by leukemia. It was also revealed that potent ***tumor***-specific, T-cell-mediated immunity was generated in mice which survived in this immunotherapy model. Thus, these results indicate that

administration of MDP ***hapten*** -modified, syngeneic leukemia cells into leukemia-bearing mice which have been primed with BCG results in potent ***tumor*** -specific, T-cell-mediated immunity attributable to preventing the growth of disseminated leukemic cells.

4/7/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

05358790 84282790
Tumor bearer T cells suppress Bacillus Calmette-Guerin-potentiated antitumor responses. III. Identification of an auxiliary efferent suppressor-T-cell population.
Hawrylko E; Mele CA
Cell Immunol (UNITED STATES) Sep 1984, 87 (2) p566-79, ISSN 0008-8749 Journal Code: CQ9
Languages: ENGLISH
Document type: JOURNAL ARTICLE
T cells (Ts-eff) induced in BALB/c mice by subcutaneous (sc) growth of syngeneic Meth A tumors can adoptively suppress the effector phase of delayed-type hypersensitivity (DTH) in Bacillus Calmette-Guerin (BCG)-primed and unprimed recipients which have been sensitized with ***irradiated*** Meth A cells but they do not inhibit the augmented DTH response in recipients inoculated with cyclophosphamide (CY) 2 days prior to sensitization. By reconstituting CY-treated immunized recipients with selected spleen cell populations, it has been demonstrated that Ts-eff suppress DTH by interacting with a second or auxiliary suppressor cell population present in immune but not normal spleens. These auxiliary suppressor cells (Ts-aux) are Thy+, Lyt 1-2+ and I-J+, phenotypically similar to Ts-eff. Their activity is not influenced by B-cell depletion. Unlike Ts-eff, Ts-aux do not bear receptors specific for Meth A cells. Ts-aux and Ts-eff share similar sensitivity to ***irradiation*** and high dose (100 mg/kg) CY but unlike Ts-eff, Ts-aux are cortisone sensitive, nondividing, nonadherent cells which are absent from the thymus. The phenotype and mechanism of action of Ts-aux resemble those of the auxiliary or Ts3 cells defined in models of contact sensitivity, DTH to simple ***haptens***, and in vitro antibody responses.

4/7/3 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

6386155 EMBASE No: 87122814
Studies on the recovery from tolerance to ***tumor*** antigens. II. Accelerated recovery of ***tumor***-specific effector T cells in tolerant mice by applying T-T cell interaction mechanism
Sato S.; Fujiwara H.; Kosugi A.; Hamaoka T.
Department of Oncogenesis, Institute for Cancer Research, Osaka University Medical School, Fukushimaku, Osaka 553 JAPAN
CANCER IMMUNOL. IMMUNOTHER. (GERMANY, WEST) , 1987, 24/2 (121-126) CODEN: CIIMD
LANGUAGES: ENGLISH
C3H/He mice were injected i.v. with heavily X-***irradiated*** syngeneic X5563 ***tumor*** cells three times at 4-day intervals. This regimen resulted in the abrogation of the potential to generate X5563 ***tumor*** -specific T cell-mediated immunity as induced by i.d. inoculation of viable X5563 ***tumor*** cells followed by surgical resection of the ***tumor***, representing the tolerance induction. Although such a ***tumor***-specific tolerant state was long-lasting, the recovery of anti-X5563 effector T cell responses was observed when the above ordinary immunization procedure was performed 6 months after the tolerance induction. The present study investigated whether the recovery from the tolerance can be accelerated by applying a helper-effector T-T cell interaction model in which enhanced anti-X5563 immunity is obtained by priming mice with BCG and by immunizing X5563 ***tumor*** cells modified with BCG cross-reactive MDP ***hapten*** (designated as L4-MDP) in the presence of anti-L4-MDP helper T cells preinduced with BCG. The results demonstrated that BCG-primed mice which received the tolerance regimen failed to generate

anti-X5563 immunity when the ordinary immunization was performed 2 or 3 months after the tolerance induction. In contrast, the immunization of BCG-primed and X5563-tolerant mice with L4-MDP-coupled X5563 ***tumor*** cells at comparable timing to that of the ordinary immunization were capable of generating potent X5563-specific in vivo protective T cell-mediated immunity. As control groups, BCG-primed or unprimed tolerant mice did not develop anti-X5563 immunity when immunized with L4-MDP-uncoupled or L4-MDP-coupled ***tumor*** cells, respectively. These results indicate that immunization of BCG-primed, ***tumor***-tolerant mice with L4-MDP-modified ***tumor*** cells results in accelerated recovery from the ***tumor*** tolerance.

4/7/4 (Item 2 from file: 73)
DIALOG(R)File 73:EMBASE

6160477 EMBASE No: 86155537
Immunological suppression in mice treated with hematoporphyrin derivative photoradiation
Elmets C.A.; Bowen K.D.
Department of Dermatology, Case Western Reserve University, Cleveland, OH 44106 USA
CANCER RES. (USA) , 1986, 46/4 (1608-1611) CODEN: CNREA LANGUAGES: ENGLISH
Hematoporphyrin derivative (HPD) is a potent photosensitizer which localized preferentially in malignant tumors. Parenteral administration of this compound followed by ***irradiation*** with the appropriate wavelengths of light has been used for the diagnosis and treatment of various epithelial neoplasms. In this study the effects of such treatment on immunological responses were evaluated by examining the capacity of HPD and light to inhibit contact hypersensitivity to dinitrofluorobenzene (DNFB) in C3H mice. Pretreatment of mice with HPD photoradiation resulted in 50% suppression of contact hypersensitivity to DNFB. Inhibition of the response could be produced even when sensitization with DNFB was attempted 2 weeks after a single ***irradiation*** procedure indicating that HPD and light-induced inhibition of contact sensitivity was a sustained phenomenon. Prior sensitization with DNFB followed by treatment with HPD and light elicited no immunosuppression. The immunosuppressive response required photoactivation of the porphyrin molecule, since mice treated with HPD alone or light alone developed little or no suppression. In adoptive transfer studies, it was shown that the immunosuppression was associated with the development of suppressor cells. These results indicate that photoradiation therapy with HPD and light can produce systemic suppression of contact hypersensitivity in mice. These data suggest that HPD photoradiation of malignant tumors may inhibit certain types of immune responses in humans.

4/7/5 (Item 3 from file: 73)
DIALOG(R)File 73:EMBASE

5594518 EMBASE No: 84090184
Studies in the enhancement of tumour immunity by coupling strong antigens to tumour cells ('heterogenization of tumours'). Helper T cell clones against PPD help other T cells mount anti-tumour responses to PPD-coupled tumour cells
Sia D.Y.; Lachmann P.J.; Leung K.N.
MRC Mechanisms in Tumour Immunity Unit, MRC Centre, Cambridge CB2 2QH UNITED KINGDOM
IMMUNOLOGY (ENGLAND) , 1984, 51/4 (755-763) CODEN: IMMUA LANGUAGES: ENGLISH
PPD-reactive T cell clones have been used to analyse the nature of T lymphocytes that are involved in the 'heterogenization' of tumour cells. This is a phenomenon where coupling tumour cells to a strong antigen (in this case PPD) causes an enhanced immune response to tumour-specific antigens to be elicited providing that the host shows T cell immunity to the strong antigen (in this case is BCG positive). Clones of T cells with the Lyt1sup +2sup - phenotype which were unable to mediate delayed-type hypersensitivity but which provided efficient help to ***hapten***-primed B cells were found to potentiate anti-tumour immunity in BCG-negative syngeneic mice when immunized with Con-A-PPD coupled, X-***irradiated*** MC6A tumour cells. There therefore appears to be a mechanism whereby a helper T cell response to one

antigen can provide help for the generation of a T cell response to a linked antigen which is analogous to the well-known phenomenon of help to ***hapten*** primed B cells. Furthermore, the clones of T cells that help B cells the best are those that give maximal augmentation of T cell immunity.

4/7/6 (Item 4 from file: 73)
DIALOG(R)File 73:EMBASE

5240184 EMBASE No: 82245919
Autologous x-***irradiated*** tumour cells and percutaneous BCG in operable lung ***cancer***
Stack B.H.R.; McSwan N.; Stirling J.M.; et al.
West. Infirm., Glasgow UNITED KINGDOM
THORAX (ENGLAND) , 1982, 37/8 (588-593) CODEN: THORA
LANGUAGES: ENGLISH
To determine the value of specific immunotherapy with adjuvant BCG in operable lung ***cancer*** , the immunological and clinical results of serial postoperative injections of autologous ***irradiated*** tumour cells and BCG were compared with those of a single preoperative injection of BCG in two randomly selected groups of patients undergoing resection of their tumours. There was a significant rise in tuberculin skin reactivity from seven weeks to 11 months after operation in the treated group. Actuarial curves for survival and freedom from tumour recurrence and median survival times showed an advantage for the treated patients who had stage I tumours, but these differences were significant only at the levels p = 0.07 - 0.09. Survival and duration of freedom from tumour recurrence was greater in autograft-treated patients whose skin responded to a weak test dose of dinitrochlorobenzene (DNCB) after sensitisation with 2% DNCB than in control DNCB-positive patients (p = 0.02). There were no significant differences in the actual proportion of patients from each group surviving at two years. The results show that this form of specific immunotherapy with adjuvant may have a beneficial effect in patients with stage I tumours and those who become sensitised to 2% DNCB after the first exposure.

4/7/7 (Item 5 from file: 73)
DIALOG(R)File 73:EMBASE

5082909 EMBASE No: 82085954
Tumor bearer T cells suppress BCG-potentiated antitumor responses. II. Characteristics of the efferent phase suppressor
Hawrylko E.; Mele C.A.; Stutman O.
Cell. Immunobiol. Sect., Mem. Sloan-Kettering Cancer Cent., New York, NY 10021 USA
CELL. IMMUNOL. (USA) , 1982, 66/1 (139-151) CODEN: CLIMB LANGUAGES: ENGLISH
T cells (Ts) from BALB/c mice bearing 6-day-old syngeneic Meth A tumors can mediate suppression of the efferent limb of the delayed-type hypersensitivity (DTH) response elicited by immunization with ***irradiated*** Meth A cells as well as the augmented response obtained when the immunogen is inoculated into BCG-primed sites. Ts in the spleen are Lyt 1sup -23sup + and bear I-J determinants. Their induction is thymus dependent. Hydrocortisone depletes the thymus of Ts but spares Ts in the spleen. Ts are metabolically active and dividing as evidenced by their sensitivity to low doses of cyclophosphamide (CY) (20 mg/kg body wt), a 15-hr pulse with vinblastine (Vbl), and ***irradiation*** with 600 rad or more. Ts adhere to Sephadex G-10 columns but are unaffected by treatment with carbonyl iron and magnetism. Their phenotype and mechanism of action appear to be comparable to those of Tssub 2 cells defined in models of DTH to simple ***haptens***.
?t s3/6/1-7

3/6/1 (Item 1 from file: 155)
06505871 88150871

The augmentation of ***tumor***-specific immunity using ***haptenic*** muramyl dipeptide (MDP) derivatives. III. Eradication of disseminated murine chronic leukemia cells by utilizing MDP ***hapten***-reactive helper T-cell activity.

3/6/2 (Item 2 from file: 155)
05358790 84282790
Tumor bearer T cells suppress Bacillus Calmette-Guerin-potentiated antitumor responses. III. Identification of an auxiliary efferent suppressor-T-cell population.

3/6/3 (Item 1 from file: 73)
7043373 EMBASE No: 88048425
The augmentation of ***tumor***-specific immunity using ***haptenic*** muramyl dipeptide (MDP) derivatives. III. Eradication of disseminated murine chronic leukemia cells by utilizing MDP ***hapten***-reactive helper T-cell activity

3/6/4 (Item 2 from file: 73)
6386155 EMBASE No: 87122814
Studies on the recovery from tolerance to ***tumor*** antigens. II. Accelerated recovery of ***tumor***-specific effector T cells in tolerant mice by applying T-T cell interaction mechanism

3/6/5 (Item 3 from file: 73)
6160477 EMBASE No: 86155537
Immunological suppression in mice treated with hematoporphyrin derivative photoradiation

3/6/6 (Item 4 from file: 73)
5699341 EMBASE No: 84195007
Tumor bearer T cells suppress Bacillus Calmette-Guerin-potentiated antitumor responses. III. Identification of an auxillary efferent suppressor-T-cell population

3/6/7 (Item 5 from file: 73)
5594518 EMBASE No: 84090184
Studies in the enhancement of tumour immunity by coupling strong antigens to tumour cells ('heterogenization of tumours'). Helper T cell clones against PPD help other T cells mount anti-tumour responses to PPD-coupled tumour cells
?t s2/6/1-7

2/6/1 (Item 1 from file: 155)
09016441 94331441
Generation of interleukin-2-secreting melanoma cell populations from resected metastatic tumors.

2/6/2 (Item 2 from file: 155)
09014528 94329528
Persistence of dormant ***tumor*** cells in the bone marrow of ***tumor*** cell-vaccinated mice correlates with long-term immunological protection.

2/6/3 (Item 3 from file: 155)
08992348 94307348
Efficacy of ***tumor*** cell ***vaccine*** after incorporating monophosphoryl lipid A (MPL) in ***tumor*** cell membranes containing ***tumor***-associated ganglioside.

2/6/4 (Item 4 from file: 155)
08983845 94298845
Induction of murine cytotoxic T lymphocytes against Plasmodium falciparum sporozoite surface protein 2.

2/6/5 (Item 5 from file: 155)
08967764 94282764
Immunotherapy of bladder ***cancer*** with cytokine gene-modified ***tumor*** ***vaccines***.

2/6/6 (Item 6 from file: 155)
08931824 94246824
Effect of perioperative chemoimmunotherapy with cyclophosphamide and autologous ***tumor*** ***vaccine*** in murine MBT-2 bladder ***cancer***.

2/6/7 (Item 7 from file: 155)
08912420 94227420
Immunotherapy of ***tumor*** metastasis via gene therapy. ?t s2/7/7

2/7/7 (Item 7 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08912420 94227420
Immunotherapy of ***tumor*** metastasis via gene therapy. Porgador A; Feldman M; Eisenbach L
Department of Cell Biology, Weizmann Institute of Science, Rehovot, Israel.
Nat Immun (SWITZERLAND) Mar-Jun 1994, 13 (2-3) p113-30, ISSN 1018-8916 Journal Code: BGD
Languages: ENGLISH
Document type: JOURNAL ARTICLE; REVIEW; REVIEW, ACADEMIC Active cellular immunotherapy of ***cancer***, via gene-modified ***tumor*** cells, may develop to an effective treatment modality. Here we discuss some of the cytokines used in gene transfer, the importance of MHC class I expression on ***tumor*** cells used as ***vaccines***, the relevance of experimental ***tumor*** systems to human tumors, and the use of live versus ***irradiated*** ***vaccines*** . Possible methods for effective gene transfer and possible protocols for immunotherapy studies are presented. Our studies in two highly metastatic murine tumors, the Lewis lung carcinoma (3LL) and the B16 melanoma, and immune mechanisms involved in immunotherapy of metastasis are summarized. (121 Refs.) ?t s2/7/6

2/7/6 (Item 6 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08931824 94246824
Effect of perioperative chemoimmunotherapy with cyclophosphamide and autologous ***tumor*** ***vaccine*** in murine MBT-2 bladder ***cancer***. Tzai TS; Huben RP; Zaleskis G; Berleth ES; Ehrke MJ; Mihich E Department of Urologic Oncology, Roswell Park Cancer Institute, Buffalo, New York 14263.

J Urol (UNITED STATES) Jun 1994, 151 (6) p1680-6, ISSN 0022-5347 Journal Code: KC7
Contract/Grant No.: CA15142, CA, NCI; CA16056, CA, NCI
Languages: ENGLISH
Document type: JOURNAL ARTICLE

The in vitro cytotoxic activity of splenocytes from C3H/He mice implanted subcutaneously with 10(6) syngeneic MBT-2 ***tumor*** cells on day 0 was significantly enhanced after cyclophosphamide (100 mg./kg., intraperitoneally) given 2 days before ***tumor*** resection on day 17, with or without active specific immunization with BCG plus autologous ***irradiated*** ***tumor*** cells (***vaccine***) 1 week after ***tumor*** resection. Furthermore, a significantly lower ***tumor*** incidence was seen in mice challenged with 10(5), but not 10(6), ***tumor*** cells per mouse 24 hours after ***tumor*** resection on day 17 and treated with cyclophosphamide on day 15 and postoperatively with ***vaccine*** than was found in nontreated ***tumor*** resected mice. Phenotypic analysis of cells from spleen showed that cyclophosphamide pretreatment and postoperative ***vaccine*** , either singly or in combination, induced a significant increase of both CD44+ memory T cells and CD11b+ myeloid/macrophage cells. Thus, in addition to a specific antitumor immune response, a nonspecific cytolytic mechanism may also play a role in the observed antitumor effect. ?e fluordinitrobenzene

Ref	Items	Index-term
E1	5	FLUORDIAGNOSTIK
E2	3	FLUORDIE
E3	0	*FLUORDINITROBENZENE
E4	1	FLUORDIZED
E5	1	FLUORDOPAN
E6	1	FLUORDOPANE
E7	7	FLUORDUSITASA
E8	1	FLUORDUSITASAVAL
E9	1	FLUORDUSITOTT
E10	30	FLUORE
E11	2	FLUOREACEIN
E12	1	FLUOREACETAMIDO

Enter P or PAGE for more
?e dinitrobenzene

Ref	Items	RT	Index-term
E1	3		DINITROBENZENAMIDE
E2	88		DINITROBENZENAMINE
E3	5523		*DINITROBENZENE
E4	1		DINITROBENZENE --DRUG COMBINATION --CB
E5	1		DINITROBENZENE --PHARMACOLOGY --PD
E6	1		DINITROBENZENE C 14
E7	1		DINITROBENZENE DERIVATIVE
E8	1		DINITROBENZENE SKIN TEST
E9	12		DINITROBENZENE SULFONATE
E10	0	1	DINITROBENZENE/1,3
E11	7		DINITROBENZENEAMINE
E12	1		DINITROBENZENEAZO

Enter P or PAGE for more
?e fluorodinitrobenzene

Ref Items RT Index-term
E1 80 FLUORODINITRO
E2 1 FLUORODINITROBENZEN
E3 276 2 *FLUORODINITROBENZENE
E4 1 FLUORODINITROBENZNE
E5 1 FLUORODINITRODIPHENYLSULFONE
E6 1 FLUORODINITROETHYL
E7 6 FLUORODINITROPHENYL
E8 3 FLUORODINITROPHENYLPYRIDOXAMINE
E9 1 FLUORODINITROPHENYLSULFONE
E10 1 FLUORODIOXABORIN
E11 2 FLUORODIOXINONES
E12 1 FLUORODIOXOHEXAHYDROXYPYRIMIDINE

Enter P or PAGE for more
?s e3 and (tumor or cancer)

Processing
276 FLUORODINITROBENZENE
756746 TUMOR
1012282 CANCER
S5 4 "FLUORODINITROBENZENE" AND (TUMOR OR CANCER) ?rd

...completed examining records
S6 4 RD (unique items)
?t s6/7/1-4

6/7/1 (Item 1 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)

10005884 BIOSIS Number: 95005884
ORIGINS OF BCG SURFACE CHARGE EFFECT OF IONIC STRENGTH AND CHEMICAL MODIFICATIONS ON ZETA POTENTIAL OF MYCOBACTERIUM-BOVIS BCG TICE SUBSTRAIN CELLS
KRISTENSEN S; TIAN Y; KLEGERMAN M E; GROVES M J
INST. TUBERCULOSIS RESEARCH, UNIV. ILLINOIS CHICAGO, 115 SOUTH SANGAMON ST., CHICAGO, ILL. 60607, USA.
MICROBIOS 70 (284-285). 1992. 185-198. CODEN: MCBIA
Full Journal Title: Microbios
Language: ENGLISH
The zeta potential of washed TiceTM substrain BCG organisms was measured over a range of ionic strengths from I = 0.005 to 0.1 M. No change in the isoelectric point of 3.4-3.7 was evident. Proteolytic enzymes (trypsin/chymotrypsin, pepsin, papain and pronase) and ***fluorodinitrobenzene*** abolished the cationic charge, suggesting that this is substantially due to amino groups associated with protein. Neither hot HCl nor cold trichloroacetic acid affected the charge, indicating that ionic groups are not associated with extractable polysaccharides. Methanolysis, treatment with HF and carbodiimide, and cationic detergent(cetyltrimethylammonium bromide) binding indicated that the negative charge was provided by carboxylic acids, phosphoesters and strong acidic groups, possibly sulphates. Standardless quantitative X-ray microanalysis revealed the presence of phosphorus and sulphur on the surface of actively growing BCG colonies.

6/7/2 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

5409605 EMBASE No: 83161428
Reactivity of ***fluorodinitrobenzene*** with intact human leucocytes: Examination of sites of action and the molecular entities involved in interaction
Joshi S.S.; Basrur V.S.; Sahasrabudhe M.B.
Cancer Res. Inst., Tata Meml. Cent., Parel, Bombay 400 012 INDIA J. BIOSCI. (INDIA) , 1982, 4/4 (441-448) CODEN: JOBSD
LANGUAGES: ENGLISH

6/7/3 (Item 2 from file: 73)
DIALOG(R)File 73:EMBASE

479408 EMBASE No: 76061556
Amino and carboxyl terminal analyses of hepatoma lactate dehydrogenase isozymes
Brummel M.C.; Carlotti R.J.; Stegink L.D.; et al.
Dept. Ped., Univ. Iowa Coll. Med., Iowa City, Ia. 52242 USA CANCER RES. (USA) , 1975, 35/5 (1278-1281) CODEN: CNREA LANGUAGES: ENGLISH
The Msub 4 isozyme of lactate dehydrogenase was purified to homogeneity from normal rat liver and from 2 Morris hepatomas (7777 and 7793). Amino terminal analyses with ***fluorodinitrobenzene*** failed to detect the presence of free amino terminal residues in each enzyme studied. Each enzyme contained between 3.7 and 4.1 moles of protein bound acetyl groups per mole of enzyme. The amino terminal peptide, characterized as N acetylalanylalanine, was isolated from Pronase digests of each isozyme preparation, and quantitative recovery experiments indicated that all acetyl residues were bound at the amino termini. Carboxyl terminal analyses demonstrated phenylalanine to be the carboxyl terminal residue in each enzyme studied. These data indicate no differences in either amino or carboxyl terminal regions of hepatoma Msub 4 isozymes compared to normal liver Msub 4 isozyme.

6/7/4 (Item 3 from file: 73)
DIALOG(R)File 73:EMBASE

169032 EMBASE No: 74173500
Some observations on the use of chemically labelled normal white blood cells as antigens in active immunization of leukaemia
Sahasrabudhe M.B.; Madyastha K.R.; Prema S.; Gollerkeri M.P. Cancer Res. Inst., Bombay INDIA
BIOMEDICINE (FRANCE) , 1974, 20/1 (31-39) CODEN: BIMDB
LANGUAGES: ENGLISH
The specificity and usefulness of chemically labeled normal white blood cells as efficient antigens for eliciting specific immune response in leukemic patients are reported. Active immunization with ***fluorodinitrobenzene*** (FDNB) labeled normal WBCs (10,000 molecules of FDNB per cell) is safe and does not cause any toxic side effects. Consistent prolongation of remission periods was demonstrated in actively immunized patients, thus establishing its usefulness and superiority over the conventional methods in destroying the residual leukemic cells. Since nonmalignant cells are used as antigen, the procedure is free from objections on ethical grounds.
?s e3 and (cyclophosphamide)

276 FLUORODINITROBENZENE
94310 CYCLOPHOSPHAMIDE

S7 0 "FLUORODINITROBENZENE" AND (CYCLOPHOSPHAMIDE) ?s dinitrobenzene and (tumor or cancer)

Processing
5523 DINITROBENZENE
756746 TUMOR
1012282 CANCER
S8 872 DINITROBENZENE AND (TUMOR OR CANCER)
?s s8 and cyclophosphamide

872 S8
94310 CYCLOPHOSPHAMIDE
S9 76 S8 AND CYCLOPHOSPHAMIDE
?s fluoro(w)dinitrobenzene

74488 FLUORO
5523 DINITROBENZENE
S10 135 FLUORO(W)DINITROBENZENE
?s s1 and (cancer or tumor)

Processing
Processing
Processing
Processing
59781 S1
1012282 CANCER
756746 TUMOR
S11 59781 S1 AND (CANCER OR TUMOR)
?s s10 and (cancer or tumor)

Processing
135 S10
1012282 CANCER
756746 TUMOR
S12 1 S10 AND (CANCER OR TUMOR)
?t s12/7/1

12/7/1 (Item 1 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)

10005884 BIOSIS Number: 95005884
ORIGINS OF BCG SURFACE CHARGE EFFECT OF IONIC STRENGTH AND CHEMICAL MODIFICATIONS ON ZETA POTENTIAL OF MYCOBACTERIUM-BOVIS BCG TICE SUBSTRAIN CELLS
KRISTENSEN S; TIAN Y; KLEGERMAN M E; GROVES M J
INST. TUBERCULOSIS RESEARCH, UNIV. ILLINOIS CHICAGO, 115 SOUTH SANGAMON ST., CHICAGO, ILL. 60607, USA.
MICROBIOS 70 (284-285). 1992. 185-198. CODEN: MCBIA
Full Journal Title: Microbios
Language: ENGLISH
The zeta potential of washed TiceTM substrain BCG organisms was measured over a range of ionic strengths from I = 0.005 to 0.1 M. No change in the isoelectric point of 3.4-3.7 was evident.

Proteolytic enzymes (trypsin/chymotrypsin, pepsin, papain and pronase) and ***fluorodinitrobenzene*** abolished the cationic charge, suggesting that this is substantially due to amino groups associated with protein. Neither hot HCl nor cold trichloroacetic acid affected the charge, indicating that ionic groups are not associated with extractable polysaccharides. Methanolysis, treatment with HF and carbodiimide, and cationic detergent(cetyltrimethylammonium bromide) binding indicated that the negative charge was provided by carboxylic acids, phosphoesters and strong acidic groups, possibly sulphates. Standardless quantitative X-ray microanalysis revealed the presence of phosphorus and sulphur on the surface of actively growing BCG colonies.
?d his

> > > 'HIS' not recognized as set or accession number
?display sets

Set Items Description
S1 59781 IRRADIAT? AND (TUMOR OR CANCER)
S2 1315 S1 AND VACCINE?
S3 9 S2 AND HAPTEN?
S4 7 RD (unique items)
S5 4 "FLUORODINITROBENZENE" AND (TUMOR OR CANCER) S6 4 RD (unique items)
S7 0 "FLUORODINITROBENZENE" AND (CYCLOPHOSPHAMIDE) S8 872 DINITROBENZENE AND (TUMOR OR CANCER)
S9 76 S8 AND CYCLOPHOSPHAMIDE
S10 135 FLUORO(W)DINITROBENZENE
S11 59781 S1 AND (CANCER OR TUMOR)
S12 1 S10 AND (CANCER OR TUMOR)
?t s9/6/1-10

9/6/1 (Item 1 from file: 155)
05408621 85024621
Potentiation of human cell-mediated and humoral immunity by low-dose ***cyclophosphamide***.

9/6/2 (Item 1 from file: 5)
10052548 BIOSIS Number: 95052548
GLUTATHIONE S-TRANSFERASE ACTIVITY AND ISOENZYME COMPOSITION IN BENIGN OVARIAN TUMOURS UNTREATED MALIGNANT OVARIAN TUMOURS AND MALIGNANT OVARIAN TUMOURS AFTER PLATINUM-***CYCLOPHOSPHAMIDE*** CHEMOTHERAPY

9/6/3 (Item 2 from file: 5)
4790764 BIOSIS Number: 79033079
POTENTIATION OF HUMAN CELL-MEDIATED AND HUMORAL IMMUNITY BY LOW-DOSE ***CYCLOPHOSPHAMIDE***

9/6/4 (Item 3 from file: 5)
4123955 BIOSIS Number: 76073806
AUGMENTATION OF THE HUMAN IMMUNE RESPONSE BY CYCLO PHOSPHAMIDE

9/6/5 (Item 1 from file: 73)
9145244 EMBASE No: 94096360
Melanoma vaccines. Current status and future prospects

9/6/6 (Item 2 from file: 73)
8974974 EMBASE No: 93278716
Treatment of human melanoma with a Hapten-modified autologous vaccine

9/6/7 (Item 3 from file: 73)
8678080 EMBASE No: 92358620
Systemic therapy in disseminated melanoma

9/6/8 (Item 4 from file: 73)
8659666 EMBASE No: 92340704
Glutathione S-transferase activity and isoenzyme composition in benign ovarian tumours, untreated malignant ovarian tumours, and malignant ovarian tumours after platinum/***cyclophosphamide*** chemotherapy

9/6/9 (Item 5 from file: 73)
8401711 EMBASE No: 92075300
Adjuvant treatment of malignant melanoma
ADJUVANTE THERAPIE DES MALIGNEN MELANOMS

9/6/10 (Item 6 from file: 73)
7791271 EMBASE No: 90224420
Antibody-directed enzyme prodrug therapy
?t s9/7/5

9/7/5 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

9145244 EMBASE No: 94096360
Melanoma vaccines. Current status and future prospects
Hersey P.
Maddison Clinical Sciences Building, Royal Newcastle Hospital, Newcastle, NSW 2300 Australia
DRUGS (New Zealand), 1994, 47/3 (373-382) CODEN: DRUGA ISSN: 0012-6667
LANGUAGES: English
?t s9/6/11-20

9/6/11 (Item 7 from file: 73)
6403584 EMBASE No: 87140245
Inhibition of suppressor T lymphocytes (Ts) by cimetidine

9/6/12 (Item 8 from file: 73)
6343683 EMBASE No: 87080339
Classification of ***cancer*** chemotherapeutic drugs according to their ability to potentiate contact sensitivity to dinitrofluorobenzene

9/6/13 (Item 9 from file: 73)
6319371 EMBASE No: 87056024
Specific immunotherapy with suppressor function inhibition for metastatic renal cell carcinoma

9/6/14 (Item 10 from file: 73)
6251820 EMBASE No: 86246883
Classification of anticancer chemotherapeutic drugs according to their immunopotentiating activity

9/6/15 (Item 11 from file: 73)
6099534 EMBASE No: 86094594
Dermatology
DERMATOLOGIE

9/6/16 (Item 12 from file: 73)
6097634 EMBASE No: 86092694
Intradermal administration of 4-hydroperoxy-***cyclophosphamide*** during contact sensitization potentiates effector T cell responsiveness in draining lymph nodes

9/6/17 (Item 13 from file: 73)
6081614 EMBASE No: 86076674
Potentiation of T-lymphocyte function by bleomycin

9/6/18 (Item 14 from file: 73)
6028862 EMBASE No: 86023922
Treatment of cutaneous T-cell lymphoma

9/6/19 (Item 15 from file: 73)
5981925 EMBASE No: 85227435
Adjuvant therapy of cutaneous malignant melanoma: A critical review

9/6/20 (Item 16 from file: 73)
5908461 EMBASE No: 85153971
Use of BCG or levamisole as an adjunct to chemotherapy or radiotherapy in malignant lymphomas
?t s9/7/14,19,21

9/7/14 (Item 10 from file: 73)
DIALOG(R)File 73:EMBASE

6251820 EMBASE No: 86246883
Classification of anticancer chemotherapeutic drugs according to their immunopotentiating activity
Ben-Efraim S.; Shoval S.; Gal T.; Ophir R.
Department of Human Microbiology, Sackler School of Medicine, Tel-Aviv University, Tel Aviv 69978 ISRAEL
ANTICANCER RES. (GREECE) , 1986, 6/4 (801-806) CODEN: ANTRD LANGUAGES: ENGLISH

9/7/19 (Item 15 from file: 73)
DIALOG(R)File 73:EMBASE

5981925 EMBASE No: 85227435

Adjuvant therapy of cutaneous malignant melanoma: A critical review Koh H.K.; Sober A.J.; Harmon D.C.; et al.
Department of Dermatology, Masssachusetts General Hospital, Boston, MA 02114 USA
MED. PEDIATR. ONCOL. (USA) , 1985, 13/5 (244-260) CODEN: MPOND LANGUAGES: ENGLISH
The emergence of revised definitions for the high-risk patient with cutaneous malignant melanoma prompts us to re-examine the current status of adjuvant therapy in this disease. We wish to address the question, 'once a cutaneous melanoma is surgically removed and the patient is currently free of disease but at high risk for metastases, what can be done to prevent recurrence'?

9/7/21 (Item 17 from file: 73)
DIALOG(R)File 73:EMBASE

5891181 EMBASE No: 85136691
Immunologic aspects of gynecologic ***cancer***
Di Saia P.J.
Department of Obstetrics and Gynecology, University of California, Irvine, Medical Center, Orange, CA USA
OBSTET. GYNECOL. SURV. (USA) , 1985, 40/3 (111-135) CODEN: OGSUA LANGUAGES: ENGLISH
In recent times immunotherapy has been approached by most scientists with skepticism. However, the term immunotherapy has been much abused and is not infrequently applied to a host of procedures which lack a clear scientific rationale and involve ill-defined products of natural origin. There are several and not mutually exclusive explanations for the lack of success within the last decade to translate immunotherapy successes in rodents the human species.
?t s9/7/20

9/7/20 (Item 16 from file: 73)
DIALOG(R)File 73:EMBASE

5908461 EMBASE No: 85153971
Use of BCG or levamisole as an adjunct to chemotherapy or radiotherapy in malignant lymphomas
Advani S.H.; Gangal S.G.; Gopal R.; et al.
Tata Memorial Hospital, Bombay INDIA
INDIAN J. MED. RES. (INDIA) , 1985, 81/3 (306-312) CODEN: IJMRA LANGUAGES: ENGLISH
Non-specific immunomodulators including BCG or levamisole have been used as an adjunct to chemotherapy or radiotherapy in both Hodgkin's disease (HD) and non-Hodgkin's lymphoma (NHL). In early stage HD, BCG and levamisole failed to show any effect over the use of radiotherapy alone. In advanced stage HD, disease-free survival as well as delayed type hypersensitivity (DTH) appeared to be better though not significantly, in patients receiving combination chemotherapy interspersed with BCG as compared to chemotherapy alone. Levamisole proved to be ineffective both in HD and NHL as an immunorestorative agent. The present study indicates that non-specific immunotherapy in malignant lymphomas, administered during the induction phase of therapy may not have the desired beneficial effect. ?s bcg or bacille(w)calmette(w)guerin

32252 BCG
974 BACILLE
4381 CALMETTE
5438 GUERIN
676 BACILLE(W)CALMETTE(W)GUERIN
S13 32341 BCG OR BACILLE(W)CALMETTE(W)GUERIN

?s s13 and (cancer or tumor) and vaccine?

Processing
32341 S13
1012282 CANCER
756746 TUMOR
188293 VACCINE?
S14 7357 S13 AND (CANCER OR TUMOR) AND VACCINE?
?s s14 and adjuvant?

7357 S14
74860 ADJUVANT?
S15 1430 S14 AND ADJUVANT?
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Set Items Description
S1 59781 IRRADIAT? AND (TUMOR OR CANCER)
S2 1315 S1 AND VACCINE?
S3 9 S2 AND HAPTEN?
S4 7 RD (unique items)
S5 4 "FLUORODINITROBENZENE" AND (TUMOR OR CANCER) S6 4 RD (unique items)
S7 0 "FLUORODINITROBENZENE" AND (CYCLOPHOSPHAMIDE) S8 872 DINITROBENZENE AND (TUMOR OR CANCER)
S9 76 S8 AND CYCLOPHOSPHAMIDE
S10 135 FLUORO(W)DINITROBENZENE
S11 59781 S1 AND (CANCER OR TUMOR)
S12 1 S10 AND (CANCER OR TUMOR)
S13 32341 BCG OR BACILLE(W)CALMETTE(W)GUERIN
S14 7357 S13 AND (CANCER OR TUMOR) AND VACCINE?
S15 1430 S14 AND ADJUVANT?
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File 5:BIOSIS PREVIEWS(R) 1969-1994/OCT W3
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E2 1 AU=BERD M
E3 0 *AU=BERD, D
E4 1 AU=BERDA C
E5 3 AU=BERDACH J T
E6 1 AU=BERDAGUE J J
E7 15 AU=BERDAGUE J L
E8 4 AU=BERDAGUE J-L
E9 12 AU=BERDAH J
E10 3 AU=BERDAH J F
E11 1 AU=BERDAH J-F
E12 3 AU=BERDAH L

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E3 83 *AU=BERD D
E4 2 AU=BERD D A
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E6 1 AU=BERDA C
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E8 1 AU=BERDAGUE J J
E9 15 AU=BERDAGUE J L
E10 4 AU=BERDAGUE J-L
E11 12 AU=BERDAH J
E12 3 AU=BERDAH J F

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83 AU=BERD D
200239 CANCER
245927 TUMOR
27781 MELANOMA
S1 63 AU="BERD D" AND (CANCER OR TUMOR OR MELANOMA) ?s s1 and dt=ab

63 S1
0 DT=AB
S2 0 S1 AND DT=AB
?s s1 and dt=abstract

63 S1
0 DT=ABSTRACT
S3 0 S1 AND DT=ABSTRACT
?t s1/6/1-63

?t s1/7/27

1/7/27
DIALOG(R)File 5:BIOSIS PREVIEWS(R)
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7297759 BIOSIS Number: 38078280
TUMOR INFILTRATING T CELLS IN METASTATIC ***MELANOMA*** INDUCTION BY IMMUNIZATION WITH AUTOLOGOUS DNP-CONJUGATED ***TUMOR*** CELLS MURPHY G F; RADU A; MASTRANGELO M; ***BERD D***
UNIV. PA., PHILADELPHIA, PA.
ANNUAL MEETING OF THE INTERNATIONAL ACADEMY OF PATHOLOGY (UNITED STATES-CANADIAN DIVISION), BOSTON, MASSACHUSETTS, USA, MARCH 4-9, 1990. LAB INVEST 62 (1). 1990. 70A. CODEN: LAINA
Language: ENGLISH
?s s1 and py=1990

63 S1
555681 PY=1990
S4 5 S1 AND PY=1990
?t s4/3/1-5

4/3/1
DIALOG(R)File 5:BIOSIS PREVIEWS(R)
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8108644 BIOSIS Number: 91029644
TREATMENT OF METASTATIC ***MELANOMA*** WITH AN AUTOLOGOUS ***TUMOR*** -CELL VACCINE CLINICAL AND IMMUNOLOGIC RESULTS IN 64 PATIENTS ***BERD D***; MAGUIRE H C JR; MCCUE P; MASTRANGELO M J
THOMAS JEFFERSON UNIV., 1015 WALNUT ST., SUITE 1005, PHILADELPHIA, PA. 19107.
J CLIN ONCOL 8 (11). 1990. 1858-1867. CODEN: JCOND
Full Journal Title: Journal of Clinical Oncology
Language: ENGLISH

4/3/2
DIALOG(R)File 5:BIOSIS PREVIEWS(R)
(c) 1994 BIOSIS. All rts. reserv.

8108551 BIOSIS Number: 91029551
A RANDOMIZED STUDY OF METHANOL-EXTRACTION RESIDUE OF BCG AS POSTSURGICAL ADJUVANT THERAPY OF UVEAL ***MELANOMA***
MCLEAN I W; ***BERD D***; MASTRANGELO M J; SHIELDS J A; DAVIDORF F H; GREVER M; MAKLEY T A; GAMEL J W
DEP. OPHTHALMIC PATHOLOGY, ARMED FORCES INST. PATHOLOGY, WASHINGTON, DC

20306-6000.
AM J OPHTHALMOL 110 (5). 1990. 522-526. CODEN: AJOPA
Full Journal Title: American Journal of Ophthalmology
Language: ENGLISH

4/3/3
DIALOG(R)File 5:BIOSIS PREVIEWS(R)

8097301 BIOSIS Number: 91018301
TIME TO RECURRENCE VARIES INVERSELY WITH THICKNESS IN CLINICAL STAGE 1 CUTANEOUS ***MELANOMA***
SCHULTZ S; KANE M; ROUSH R; MILLER V; ***BERD D***; GOLDMAN L; MASTRANGELO M
DEP. MED., DIV. MED. ONCOL., JEFFERSON MED. COLL., 1025 WALNUT ST., PHILADELPHIA, PA. 19107.
SURG GYNECOL OBSTET 171 (5). 1990. 393-397. CODEN: SGOBA Full Journal Title: Surgery Gynecology & Obstetrics
Language: ENGLISH

4/3/4
DIALOG(R)File 5:BIOSIS PREVIEWS(R)

7530160 BIOSIS Number: 39042767
LYMPHOCYTIC INFILTRATION OF ***MELANOMA*** METASTASES INDUCED BY IMMUNIZATION WITH DINITROPHENYL DNP-CONJUGATED ***TUMOR*** CELLS ***BERD D***; MAGUIRE H C JR; MASTRANGELO M J; MURPHY G
THOMAS JEFFERSON UNIV., PHILADELPHIA, PA.
81ST ANNUAL MEETING OF THE AMERICAN ASSOCIATION FOR CANCER RESEARCH, WASHINGTON, D.C., USA, MAY 23-26, 1990. PROC AM ASSOC CANCER RES ANNU MEET 31 (0). 1990. 279. CODEN: PAMRE
Language: ENGLISH
Document Type: CONFERENCE PAPER

4/3/5
DIALOG(R)File 5:BIOSIS PREVIEWS(R)

7297759 BIOSIS Number: 38078280
TUMOR INFILTRATING T CELLS IN METASTATIC ***MELANOMA*** INDUCTION BY IMMUNIZATION WITH AUTOLOGOUS DNP-CONJUGATED ***TUMOR*** CELLS MURPHY G F; RADU A; MASTRANGELO M; ***BERD D***
UNIV. PA., PHILADELPHIA, PA.
ANNUAL MEETING OF THE INTERNATIONAL ACADEMY OF PATHOLOGY (UNITED STATES-CANADIAN DIVISION), BOSTON, MASSACHUSETTS, USA, MARCH 4-9, 1990. LAB INVEST 62 (1). 1990. 70A. CODEN: LAINA
Language: ENGLISH
Document Type: CONFERENCE PAPER
?s s1 and py=1989

63 S1

514758 PY=1989
S5 6 S1 AND PY=1989
?s s5 and dt=conference paper

6 S5
2347102 DT=CONFERENCE PAPER
S6 2 S5 AND DT=CONFERENCE PAPER
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6/7/1
DIALOG(R)File 5:BIOSIS PREVIEWS(R)

6869219 BIOSIS Number: 37063598
TUMOR INFLAMMATORY RESPONSE INDUCED BY IMMUNIZATION WITH AUTOLOGOUS ***MELANOMA*** CELLS CONJUGATED TO DINITROPHENOL DNP
BERD D; MASTRANGELO M J; GREEN C; CLARK C; HART E
THOMAS JEFFERSON UNIV., PHILADELPHIA, PA. 19107, USA.
EIGHTIETH ANNUAL MEETING OF THE AMERICAN ASSOCIATION FOR CANCER RESEARCH, SAN FRANCISCO, CALIFORNIA, USA, MAY 24-27, 1989. PROC AM ASSOC CANCER RES ANNU MEET 30 (0). 1989. 382. CODEN: PAMRE
Language: ENGLISH

6/7/2
DIALOG(R)File 5:BIOSIS PREVIEWS(R)

6810858 BIOSIS Number: 37005237
IMMUNOTHERAPY OF ***MELANOMA*** WITH AUTOLOGOUS ***TUMOR*** VACCINE PRECEDED BY LOW DOSE CYCLOPHOSPHAMIDE
BERD D
DIV. MED. ONCOL., THOMAS JEFFERSON UNIV., PHILADELPHIA, PA. 19107. METZGAR, R. S. AND M. S. MITCHELL (ED.). UCLA (UNIVERSITY OF CALIFORNIA-LOS ANGELES) SYMPOSIA ON MOLECULAR AND CELLULAR BIOLOGY NEW SERIES, VOL. 99. HUMAN TUMOR ANTIGENS AND SPECIFIC TUMOR THERAPY; KEYSTONE, COLORADO, USA, APRIL 23-30, 1988. XIX+366P. ALAN R. LISS, INC.: NEW YORK, NEW YORK, USA. ILLUS. ISBN 0-8451-2698-9. 0 (0). 1988 (1989). 297-306. CODEN: USMBD
Language: ENGLISH
?t s5/6/1-6

5/6/1
7388553 BIOSIS Number: 89039572
FLOW CYTOMETRIC DETERMINATION OF THE FREQUENCY AND HETEROGENEITY OF EXPRESSION OF HUMAN ***MELANOMA***-ASSOCIATED ANTIGENS

5/6/2
7388545 BIOSIS Number: 89039564
DEPLETION OF T-CELLS WITH THE CD4-POSITIVE CD45R-POSITIVE PHENOTYPE IN LYMPHOCYTES THAT INFILTRATE SUBCUTANEOUS METASTASES OF HUMAN ***MELANOMA***

5/6/3
7152922 BIOSIS Number: 88075667
FLOW CYTOMETRIC EVALUATION OF BRONCHOSCOPIC WASHINGS AND LAVAGE FLUID FOR DNA ANEUPLOIDY AS AN ADJUNCT IN THE DIAGNOSIS OF LUNG ***CANCER*** AND TUMORS METASTATIC TO THE LUNG

5/6/4
7056860 BIOSIS Number: 87117381
THE IMPORTANCE OF TAMOXIFEN TO A CISPLATIN-CONTAINING REGIMEN IN THE TREATMENT OF METASTATIC ***MELANOMA***

5/6/5
6869219 BIOSIS Number: 37063598
TUMOR INFLAMMATORY RESPONSE INDUCED BY IMMUNIZATION WITH AUTOLOGOUS ***MELANOMA*** CELLS CONJUGATED TO DINITROPHENOL DNP

5/6/6
6810858 BIOSIS Number: 37005237
IMMUNOTHERAPY OF ***MELANOMA*** WITH AUTOLOGOUS ***TUMOR*** VACCINE PRECEDED BY LOW DOSE CYCLOPHOSPHAMIDE
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=> e fluordinitrobenzene/cn

E1 1 FLUORCLINOHUMITE/CN
E2 1 FLUORCLINOHUMITE ((MG0.5-1FE0-0.5)9(F0.5-1(OH)0-0.5)2(SIO4)4)/CN
E3 0 --> FLUORDINITROBENZENE/CN
E4 1 FLUORECEIN, 2',4',5',7'-TETRABROMO-, COMPD. WITH 4,6-DIAMINO-1-(P-CHLOROPHENYL)-1,2-DIHYDRO-2,2-DIMETHYL-S-TRIAZINE/CN
E5 1 FLUORECITE/CN
E6 1 FLUOREDENITE (NA(ALCA2MG5F2O(SIO3)7))/CN E7 1 FLUOREKS/CN
E8 1 FLUOREKS 1510/CN
E9 1 FLUORELLESTADITE/CN
E10 1 FLUORELLESTADITE (CA5(F0.5-1CL0-0.5(OH)0-0.5)(((SIO4)0.5(SO4)0.5)0.5-1(PO4)0-0.5)3)/CN
E11 1 FLUOREMBICHIN/CN
E12 1 FLUOREN-1(2H)-ONE, 2-IMINO-/CN

=> e fluorodinitrobenzene/cn

E1 1 FLUORODIMETHYLSILYL ISOTHIOCYANATE/CN
E2 1 FLUORODINITROACETONITRILE/CN
E3 2 --> FLUORODINITROBENZENE/CN
E4 1 FLUORODINITROETHYL (PENTAFLUOROTHIO)ACETATE/CN E5 1 FLUORODINITROMETHANE/CN
E6 1 FLUORODINITROMETHANE ANION/CN
E7 1 FLUORODINITROMETHYL ANION/CN
E8 1 FLUORODINITROPHENYLMETHANE/CN
E9 1 FLUORODIOXOBISPYRIDINERHENIUM/CN
E10 1 FLUORODIOXOTRISPYRIDINERHENIUM/CN
E11 1 FLUORODIOXOXENON(1+) HEXAFLUOROARSENATE(1-)/CN E12 1 FLUORODIOXY/CN

=> s e3

L1 2 FLUORODINITROBENZENE/CN

=> s e3:d

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=> s e3; d

L3 2 FLUORODINITROBENZENE/CN

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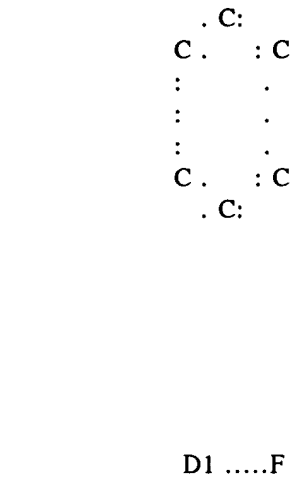
RN 25376-51-6 REGISTRY
CN Benzene, fluorodinitro- (8CI, 9CI) (CA INDEX NAME)
OTHER NAMES:
CN ***Fluorodinitrobenzene***
MF C6 H3 F N2 O4
CI IDS
LC STN Files: BIOBUSINESS, BIOSIS, CA, CAPREVIEWS, EMBASE, IFICDB, IFIPAT, IFIUDB, TOXLIT
DES 8:ID



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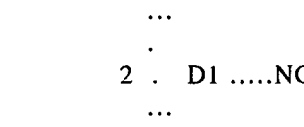

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76 25376-51-6/BI
L5 147 (FLUORODINITROBENZENE/BI OR 25376-51-6/BI)

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L6 172 (FLUORODINITROBENZENE/BI OR 25376-51-6/BI)

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=> s l7 and (cancer or tumor)

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48705 CANCER
106063 TUMOR
L8 3 L4 AND (CANCER OR TUMOR)

FILE 'BIOSIS'
200255 CANCER
244258 TUMOR

L9 1 L5 AND (CANCER OR TUMOR)

FILE 'EMBASE'
346042 CANCER
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L12 20 DUP REM L11 (0 DUPLICATES REMOVED)

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L12 ANSWER 2 OF 20 EMBASE COPYRIGHT 1994 ELSEVIER SCI. B.V. TI Induction of hapten-specific tolerance by interleukin 10 in vivo.
L12 ANSWER 3 OF 20 EMBASE COPYRIGHT 1994 ELSEVIER SCI. B.V. TI Cytokine induction in human epidermal keratinocytes exposed to contact irritants and its relation to chemical-induced inflammation in mouse skin.

L12 ANSWER 4 OF 20 EMBASE COPYRIGHT 1994 ELSEVIER SCI. B.V. TI Characterization of the immunogenetic basis of ultraviolet-B light effects on contact hypersensitivity induction.

L12 ANSWER 5 OF 20 EMBASE COPYRIGHT 1994 ELSEVIER SCI. B.V. TI Evidence that ultraviolet B radiation induces tolerance and impairs induction of contact hypersensitivity by different mechanisms.
L12 ANSWER 6 OF 20 EMBASE COPYRIGHT 1994 ELSEVIER SCI. B.V. TI The effect of anti-***tumor*** necrosis factor (TNF)-.alpha. monoclonal antibody on allergic cutaneous late phase reaction in mice.

L12 ANSWER 7 OF 20 EMBASE COPYRIGHT 1994 ELSEVIER SCI. B.V. TI Depletion of Langerhans cells following carcinogen treatment is partly due to antigenicity.

L12 ANSWER 8 OF 20 EMBASE COPYRIGHT 1994 ELSEVIER SCI. B.V. TI Pentoxifylline suppresses irritant and contact hypersensitivity reactions.

L12 ANSWER 9 OF 20 EMBASE COPYRIGHT 1994 ELSEVIER SCI. B.V. TI Suppression of the delayed type hypersensitivity response by ***tumor*** facilitating factor of B16 melanoma: A ***tumor*** factor suppresses immune responses.

L12 ANSWER 10 OF 20 EMBASE COPYRIGHT 1994 ELSEVIER SCI. B.V. TI Treatment of human melanoma with a Hapten-modified autologous vaccine.

L12 ANSWER 11 OF 20 EMBASE COPYRIGHT 1994 ELSEVIER SCI. B.V. TI cis-Urocanic acid suppression of contact hypersensitivity induction is mediated via ***tumor*** necrosis factor-.alpha..
L12 ANSWER 12 OF 20 BIOSIS COPYRIGHT 1994 BIOSIS
TI ORIGINS OF BCG SURFACE CHARGE EFFECT OF IONIC STRENGTH AND CHEMICAL MODIFICATIONS ON ZETA POTENTIAL OF MYCOBACTERIUM-BOVIS BCG TICE SUBSTRAIN CELLS.

L12 ANSWER 13 OF 20 EMBASE COPYRIGHT 1994 ELSEVIER SCI. B.V. TI Local- and systemic-mediated suppression of contact hypersensitivity in mice by several structurally unrelated classes of ***tumor*** promoters.

L12 ANSWER 14 OF 20 EMBASE COPYRIGHT 1994 ELSEVIER SCI. B.V. TI Genetic basis of the effects of ultraviolet light B on cutaneous immunity. Evidence that polymorphism at the Tnfa and Lps loci governs susceptibility.

L12 ANSWER 15 OF 20 EMBASE COPYRIGHT 1994 ELSEVIER SCI. B.V. TI Sensitization through carcinogen-induced Langerhans cell-deficient skin activates specific long-lived suppressor cells for both cellular and humoral immunity.

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TI Determination of galactosyl-N-acetylglucosamine and galactosyl-N-acetylgalactosamine-containing substances in body fluids for diagnosis of ***cancer***

L12 ANSWER 17 OF 20 EMBASE COPYRIGHT 1994 ELSEVIER SCI. B.V. TI Amino and carboxyl terminal analyses of hepatoma lactate dehydrogenase isozymes.

L12 ANSWER 18 OF 20 EMBASE COPYRIGHT 1994 ELSEVIER SCI. B.V. TI Some observations on the use of chemically labelled normal white blood cells as antigens in active immunization of leukaemia.
L12 ANSWER 19 OF 20 CA COPYRIGHT 1994 ACS
TI Immunization with chemically modified lymphoma cells

L12 ANSWER 20 OF 20 CA COPYRIGHT 1994 ACS
TI ***Tumor*** promotion by 1-fluoro-2,4-dinitrobenzene, a potent skin sensitizer

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L12 ANSWER 19 OF 20 CA COPYRIGHT 1994 ACS
AN 76:21542 CA
TI Immunization with chemically modified lymphoma cells
AU Prager, Morton D.; Derr, Ina; Swann, Alan; Cotropia, Joseph CS Southwest. Med. Sch., Univ. Texas, Dallas, Tex., USA
SO Cancer Res. (1971), 31(10), 1488-91
CODEN: CNREA8
DT Journal
LA English

L12 ANSWER 20 OF 20 CA COPYRIGHT 1994 ACS
AN 70:66204 CA
TI ***Tumor*** promotion by 1-fluoro-2,4-dinitrobenzene, a potent skin sensitizer
AU Bock, Fred G.; Fjelde, Audrey; Fox, Helen W.; Klein, Edmund CS Roswell Park Mem. Inst., Buffalo, N. Y., USA
SO Cancer Res. (1969), 29(1), 179-82
CODEN: CNREA8

DT Journal
LA English

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L12 ANSWER 19 OF 20 CA COPYRIGHT 1994 ACS
AN 76:21542 CA
TI Immunization with chemically modified lymphoma cells
AU Prager, Morton D.; Derr, Ina; Swann, Alan; Cotropia, Joseph CS Southwest. Med. Sch., Univ. Texas, Dallas, Tex., USA
SO Cancer Res. (1971), 31(10), 1488-91
CODEN: CNREA8
DT Journal
LA English
AB Immunization of C3H mice with 6C3HED ascites lymphosarcoma cells modified with the strong antigenic determinants, diazotized p-aminobenzoic acid [150-13-0] or ***fluorodinitrobenzene*** [70-34-8], failed to protect the animals against a challenging lethal dose of the ***tumor*** cells; iodoacetamide (I) [144-48-9], iodoacetate [64-69-7], and N-ethylmaleimide [128-53-0] protected fully; p-hydroxymercuribenzoate [1126-48-3] gave partial protection. Alterations producing potent immunity involved blocking sulfhydryl groups with reagents not generally considered good haptens. I-treated EPF-1 lymphoma protected against an early transplant generation of EPF-1. No cytotoxic antibody to 6C3HED or EPF-1 in immune serum was demonstrable, but lymphoid cells from an immune C3H mouse protected a susceptible animal, indicating cell-mediated immunity.

L12 ANSWER 20 OF 20 CA COPYRIGHT 1994 ACS
AN 70:66204 CA
TI ***Tumor*** promotion by 1-fluoro-2,4-dinitrobenzene, a potent skin sensitizer
AU Bock, Fred G.; Fjelde, Audrey; Fox, Helen W.; Klein, Edmund CS Roswell Park Mem. Inst., Buffalo, N. Y., USA
SO Cancer Res. (1969), 29(1), 179-82
CODEN: CNREA8
DT Journal
LA English
AB 1-Fluoro-2,4-dinitrobenzene (I) was a potent ***tumor*** -promoting agent. After topical application of 7,12- dimethylbenz[a]anthracene (125 .mu.g.) followed by 5 paintings a week with 0.1% I in Me2CO, mice developed tumors in 4 weeks. After 38 weeks, .apprx.67% of the animals had tumors. Treatment with I alone did not produce tumors, nor was N6-(2,4-dinitrophenyl)-L- lysine-HCl, the lysine conjugate of I, a ***tumor*** promoter. A relation between ***tumor*** promotion and disturbance of the immune system in sensitive animals is suggested.

=> d l12 9,13 bib ab

L12 ANSWER 9 OF 20 EMBASE COPYRIGHT 1994 ELSEVIER SCI. B.V. AN 94179484 EMBASE
TI Suppression of the delayed type hypersensitivity response by ***tumor*** facilitating factor of B16 melanoma: A ***tumor*** factor suppresses immune responses.
AU Burnstein R.; Brody N.I.
CS Department of Dermatology, Box 46, SUNY, 450 Clarkson Ave, Brooklyn, NY 11203, United States
SO J. DERMATOL. SURG. ONCOL., (1993) 19/6 (543-552).
ISSN: 0148-0812 CODEN: JDSODJ
CY United States
DT Journal
FS 013 Dermatology and Venereology

016 Cancer
026 Immunology, Serology and Transplantation
029 Clinical Biochemistry
LA English
SL English
AB BACKGROUND. ***Tumor*** facilitating factor is a cell surface glycoprotein produced by B16 melanoma that has been found to reduce the lethal inoculum for B16. ***Tumor*** facilitating factor induces macrophage spreading in vitro, reduces macrophage chemotaxis in vivo, and depresses lymphocyte mitogenesis in vitro. OBJECTIVE. It is assumed that the immune modifying effects are responsible for ***tumor*** facilitation. As tumors may be poor immunogens or inducers of inflammation, studies were conducted to determine whether ***tumor*** facilitating factor alters the inflammatory cascade of cells found in infiltrates of delayed type hypersensitivity. RESULTS. Freeze-thawed B16 cells, used as the source of TFF, caused a suppression of delayed type hypersensitivity measured as ear swelling in the mouse. When culture supernatant was substituted for freeze-thawed cells as a source of TFF and injected at different time points of the delayed type hypersensitivity response, the greater suppression was with ***tumor*** facilitating factor injections at 24 hours pre-elicitation only (82%), and 24 hours both presensitization and pre-elicitation (89%). Immunohistological staining demonstrated that ***tumor*** facilitating factor decreases ear thickness and cellular infiltrates, specifically Mac-1 staining cells, to a site of delayed hypersensitivity. Peritoneal cell analysis confirmed these findings. CONCLUSION. These data are consistent with the hypothesis that ***tumor*** facilitating factor alters immune functions including macrophage and lymphocyte mobility and recruitment to a target site, thereby allowing for facilitation of ***tumor*** growth.
L12 ANSWER 13 OF 20 EMBASE COPYRIGHT 1994 ELSEVIER SCI. B.V. AN 91297297 EMBASE
TI Local- and systemic-mediated suppression of contact hypersensitivity in mice by several structurally unrelated classes of ***tumor*** promoters.
AU Kodari E.; Pavone A.; Reiners J.J. Jnr.
CS Department of Carcinogenesis, Science Park-Research Division, University of Texas M.D. Anderson Cancer Center, Smithville, TX 78957, United States
SO CARCINOGENESIS, (1991) 12/10 (1933-1937).
ISSN: 0143-3334 CODEN: CRNGDP
CY United Kingdom
DT Journal
FS 016 Cancer
LA English
SL English
AB Several structurally unrelated classes of chemicals defined as promoters in the murine skin multistage carcinogenesis protocol were surveyed for their abilities to modify contact hypersensitivity (CHS) responses in SENCAR mice. Sensitization of dorsal skin with 2,4-dinitrotluorobenzene (DNFB) and subsequent challenge of ears 5 days later with DNFB resulted within 24 h in ear swelling. Pretreatment of dorsal skin with multiple applications (2 x /week for 2 weeks) of promoting doses of 12-O-tetradecanoylphorbol-13- acetate (TPA), anthralin, butylated hydroxytoluene hydroperoxide, n-dodecane and ethyl phenylpropiolate (EPP) prior to sensitization with DNFB inhibited, to a comparable extent, the subsequent induction of CHS by DNFB challenge. Pretreatment of dorsal skin with promoting doses of benzoyl peroxide resulted in reproducible, but diminished suppression of CHS, relative to that mediated by the other chemical promoters. Application of promoting doses of TPA, anthralin and EPP, but not the other chemicals, to ventral skin prior to DNFB sensitization of dorsal skin also significantly inhibited DNFB-induced CHS. However, suppression of CHS mediated by ventral application of these three chemicals was quantitatively less than that occurring when the chemicals were applied to the site of DNFB sensitization. Collectively, these studies demonstrate that various classes of structurally unrelated ***tumor*** promoters have in common the ability to suppress CHS, a cell-mediated immune response. Furthermore, some ***tumor*** promoters exert their suppressive effects through both local and systemic processes.
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SYSTEM:OS - DIALOG OneSearch
File 155:MEDLINE(R) 1966-1994/Nov W4
(c) format only 1994 Dialog Info.Svcs.
File 5:BIOSIS PREVIEWS(R) 1969-1994/OCT W3
(c) 1994 BIOSIS
File 73:EMBASE 1974-1994/ISS 39
(c) 1994 Elsevier Science B.V.
*File 73: Truncate EMTREE codes(e.g. DC=C1.120?) for complete retrieval. See HELP NEWS 73 for explode feature.

Set Items Description
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82302 MELANOMA
431726 DT=REVIEW
S1 2310 MELANOMA AND DT=REVIEW
?s (tumor or cancer) and vaccine? and dt=review

Processing
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1012282 CANCER
188293 VACCINE?
431726 DT=REVIEW
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431726 DT=REVIEW
S3 0 MELANOMA\TI AND DT=REVIEW
?s melanoma/ti and dt=review

42294 MELANOMA/TI
431726 DT=REVIEW
S4 967 MELANOMA/TI AND DT=REVIEW
?s s4 and vaccine?

967 S4
188293 VACCINE?
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...examined 50 records (200)
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...examined 50 records (350)

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...completed examining records
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?t s5/7/33

5/7/33 (Item 33 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

03005630 76186630
The immune response in human malignant ***melanoma***.
Mackie RM
Clin Exp Dermatol (ENGLAND) Mar 1976, 1 (1) p23-8, ISSN 9995-6699 Journal Code: DDU
Languages: ENGLISH
Document type: JOURNAL ARTICLE; ***REVIEW***
(30 Refs.)
?display sets

Set Items Description
S1 2310 MELANOMA AND DT=REVIEW
S2 446 (TUMOR OR CANCER) AND VACCINE? AND DT=REVIEW S3 0
MELANOMA\TI AND DT=REVIEW
S4 967 MELANOMA/TI AND DT=REVIEW
S5 42 S4 AND VACCINE?
S6 42 RD (unique items)
S7 442 RD S2 (unique items)
?s s7/6/1-50

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?t s7/6/1-50

?t s7/7/3,8,19,41,48

7/7/3 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08957887 94272887
New possibilities for ***cancer*** therapy with advances in ***cancer*** immunology.
MacLean GD; Longenecker BM
Department of Oncology, Faculty of Medicine, University of Alberta, Edmonton.
Can J Oncol (CANADA) Apr 1994, 4 (2) p249-54, ISSN 1183-2509 Journal Code: B01
Languages: ENGLISH
Document type: JOURNAL ARTICLE; ***REVIEW***; REVIEW, TUTORIAL There has been progress over the last decade in addressing three questions: Are there ***cancer***-associated antigens that could be targets for immunotherapy? Can the human immune system recognize ***cancer*** -associated antigens? Can an anti-***cancer*** immune response affect ***cancer*** cells and lead to increased survival?

Results from animal model studies have been interpreted by optimists as encouraging, and by pessimists as being irrelevant to human ***cancer***. Earlier studies on " ***cancer*** ***vaccines*** " utilized heterogeneous cell extracts of cell components. Monoclonal antibodies have enabled identification of relevant ***cancer*** -associated antigens or epitopes, such as the ganglioside GM2, the carbohydrates TF and STn, and the peptide sequences of MUC-1. In parallel with research on immune adjuvants and measures designed to inhibit suppressor activity, these epitopes are being tested for their potential in the immunotherapy of solid tumors. It is clear that some of these ***cancer***-associated epitopes are immunogenic in humans. Mixed responses may relate to ***cancer*** heterogeneity and may indicate the importance of multi-epitopic ***vaccines*** . Responses are encouraging, but are they relevant? Prolonged disease stability challenges us to re-think the goals of ***cancer*** therapy. Recent advances in the knowledge of the effect of cytokines on ***tumor*** antigen expression and the regulation of the immune response, coupled with advances in active specific immunotherapy, provide hope that biomodulation may become an important part of the therapy of solid tumors in the next century. (32 Refs.)

7/7/8 (Item 8 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08943817 94258817
Tumor-specific immune responses and opportunities for ***tumor*** ***vaccines***.
Finn OJ
Department of Molecular Genetics and Biochemistry, University of Pittsburgh School of Medicine, Pennsylvania 15261.
Clin Immunol Immunopathol (UNITED STATES) Jun 1994, 71 (3) p260-2, ISSN 0090-1229 Journal Code: DEA
Languages: ENGLISH
Document type: JOURNAL ARTICLE; ***REVIEW***; REVIEW, TUTORIAL (25 Refs.)

7/7/19 (Item 19 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08887952 94202952
Human ***cancer*** ***vaccines***.
Sinkovics J; Horvath J; Szabo-Szabari M
Cancer Institute, St. Joseph's Hospital, Tampa, Florida 33607. Leukemia (ENGLAND) Apr 1994, 8 Suppl 1 pS194-7, ISSN 0887-6924 Journal Code: LEU
Languages: ENGLISH
Document type: JOURNAL ARTICLE; ***REVIEW***; REVIEW, TUTORIAL Immune T cells recognize peptide antigens presented to them within self-MHC molecules. Thus auto-***tumor*** reactive lymphocyte populations can be generated. Antigenic expression can be modified and intensified and reactive lymphocyte populations can be expanded. Active immunization of the ***tumor***-bearing human host can induce immune reactions of ***tumor*** rejection strength. Frequently, micrometastases can be eliminated and occasionally partial or complete remissions of gross metastases can be induced. (22 Refs.)

7/7/41 (Item 41 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08710292 94025292
Cancer ***vaccines***.

Pardoll DM
Department of Oncology, Johns Hopkins University School of Medicine, Baltimore, MD 21205.
Trends Pharmacol Sci (ENGLAND) May 1993, 14 (5) p202-8, ISSN 0165-6147 Journal Code: WFT
Languages: ENGLISH
Document type: JOURNAL ARTICLE; ***REVIEW***; REVIEW, TUTORIAL 1993 represents the 100th anniversary of William Coley's first report of tumour regressions induced by immune system activation in response to bacterial toxins. While many subsequent ***cancer*** ***vaccine*** trials have yielded tantalizing results, active immunotherapy has not yet become an established modality of ***cancer*** therapy. Drew Pardoll reviews newer molecular ***vaccine*** approaches based on rational immunological principles that have resulted in improved systemic antitumour effects in animal models. Ultimately the genetic definition of tumour-specific antigens will allow the development of targeted antigen-specific ***vaccines*** for ***cancer*** therapy. (54 Refs.)

7/7/48 (Item 48 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

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08668327 93378327
Cancer ***vaccines***: the perspective of the ***Cancer*** Immunology Branch, NCI.
Sogn JA; Finerty JF; Heath AK; Shen GL; Austin FC
Cancer Immunology Branch, National Cancer Institute, NIH, Bethesda, Maryland 20892.
Ann N Y Acad Sci (UNITED STATES) Aug 12 1993, 690 p322-30, ISSN 0077-8923 Journal Code: 5NM
Languages: ENGLISH
Document type: JOURNAL ARTICLE; ***REVIEW***; REVIEW, TUTORIAL The ***Cancer*** Immunology Branch, NCI, is supporting a great deal of exciting research relevant to ***cancer*** ***vaccine*** development. The few areas highlighted here are representative of ongoing research opportunities, but further progress depends largely on a continued infusion of investigator-initiated ideas to realize the potential of current research areas and open new ones. (22 Refs.)
?t s7/6/51-100

?t s7/7/67,83

7/7/67 (Item 67 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

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08552434 93262434
Identifying strategies for immune intervention.
Lanzavecchia A
Basel Institute for Immunology, Switzerland.
Science (UNITED STATES) May 14 1993, 260 (5110) p937-44, ISSN 0036-8075 Journal Code: UJ7
Languages: ENGLISH
Document type: JOURNAL ARTICLE; ***REVIEW***; REVIEW, TUTORIAL In recent years the molecular basis of antigen recognition by T cells has been unraveled and the various pathways that control T cell activation and functional specialization have been defined. Consequently, it is now possible to delineate various strategies for intervention with the immune system to design protective ***vaccines***, to induce an effective response to ***tumor*** antigens, and to control graft rejection and autoimmune diseases. (121 Refs.)

7/7/83 (Item 83 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08387061 93097061
Biology of metastasis: clinical implications.
Liu BC; Weiss RE; Gordon JN; Droller MJ
Department of Urology, Mount Sinai School of Medicine, New York, New York 10029.
Semin Surg Oncol (UNITED STATES) Sep-Oct 1992, 8 (5) p267-73, ISSN 8756-0437 Journal Code: SSO
Languages: ENGLISH
Document type: JOURNAL ARTICLE; ***REVIEW***; REVIEW LITERATURE Bladder ***tumor*** has a spectrum of neoplastic activity. Some behave in a benign fashion, and others are highly aggressive and lead rapidly to metastatic disease and death. The processes of metastasis can be described as a sequence of interrelated steps. The processes involve 1) ***tumor*** cell adhesion to basement membranes, 2) the degradation of basement membranes, and 3) the migration of ***tumor*** cells through the destroyed stroma into blood and lymphatic vessels. Each of these processes involves the expression of molecular factors unique to ***tumor*** cells. With better understanding of the molecular basis of these factors, novel prognostic and potential therapeutic agents can be generated and applied to the clinical arena. (34 Refs.)
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S1 2310 MELANOMA AND DT=REVIEW
S2 446 (TUMOR OR CANCER) AND VACCINE? AND DT=REVIEW S3 0
MELANOMA\TI AND DT=REVIEW
S4 967 MELANOMA/TI AND DT=REVIEW
S5 42 S4 AND VACCINE?
S6 42 RD (unique items)
S7 442 RD S2 (unique items)
?t s7/7/199,189,161,155,148,121,118

7/7/199 (Item 199 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07442653 90349653
Cancer immunotherapy with autologous and allogeneic ***vaccines***: a practical overview.
Shinitzky M; Skornick Y
Department of Membrane Research, Weizmann Institute of Science, Rehovot, Israel.
Prog Clin Biol Res (UNITED STATES) 1990, 348 p95-125, ISSN 0361-7742 Journal Code: PZ5
Languages: ENGLISH
Document type: JOURNAL ARTICLE; ***REVIEW***; REVIEW, TUTORIAL (79 Refs.)

7/7/189 (Item 189 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07485587 91004587
Tumor ***vaccines***.
Bystryn JC

Melanoma Program, Kaplan Cancer Center, New York University School of Medicine, NY 10016.
Cancer Metastasis Rev (UNITED STATES) Jul 1990, 9 (1) p81-91, ISSN 0891-9992 Journal Code: C9H
Contract/Grant No.: CA34358, CA, NCI
Languages: ENGLISH
Document type: JOURNAL ARTICLE; ***REVIEW***; REVIEW, TUTORIAL Melanoma ***vaccines*** are an exciting and increasingly attractive immunotherapeutic approach for malignant melanoma. ***Vaccines*** can be used for patients with high risk primary melanoma and regional disease, stages in the progression of melanoma for which there is presently no treatment. They are unique in their potential to prevent ***cancer*** in high risk individuals. Multiple approaches are being followed to develop effective ***vaccines*** . It is too early to judge whether any of them effectively slow the progression of melanoma. However, it is clear that ***vaccines*** are safe to use, and that they can stimulate immune responses to melanoma in some patients. The specificity of these responses needs to be clarified, and multiple challenges remain to be overcome before effective ***vaccines*** to melanoma become available. We must first identify the antigens on melanoma that stimulate immune responses, define the immune effector mechanisms that are stimulated by ***vaccine*** immunization and identify those responsible for increasing resistance to ***tumor*** growth, devise appropriate ways of constructing ***vaccines*** that will induce such responses, and find adjuvants and/or immunodulators that will potentiate desirable immune responses. (44 Refs.)

7/7/161 (Item 161 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07798389 91317389
Tumor ***vaccines***.
Stevenson FK
Tenovus Laboratory, Southampton General Hospital, United Kingdom. FASEB J (UNITED STATES) Jun 1991, 5 (9) p2250-7, ISSN 0892-6638 Journal Code: FAS
Languages: ENGLISH
Document type: JOURNAL ARTICLE; ***REVIEW***; REVIEW, TUTORIAL Vaccination against ***tumor***, either as a prophylactic procedure or as a mode of treatment, has been a distant goal of immunologists for many years. Ideally, the less specific therapies such as chemotherapy would be replaced by an anti-***tumor*** immune response in the host that would be present on a continuing basis. However, progress has been hampered by a lack of understanding of the role of viruses in human ***tumor*** development and the molecular nature of ***tumor***-associated antigens. Recent developments using the techniques of molecular biology and monoclonal antibody reagents are beginning to remedy this deficiency so that vaccination has become a real possibility for certain human cancers. The natural fluctuations in growth rates of some human tumors, and the observation that tumors can occasionally remain dormant for years, has led to the idea that the host has an intrinsic ability to control ***tumor*** growth, and that this ability is a property of the immune system. Attempts to enhance this putative control are being made by treating the host with defined biological modifiers that stimulate cells involved in immunity in vivo, and by seeking and expanding such cells in vitro before reinfusing them into the host. These attempts to harness the immune system to attack ***tumor*** cells that have evaded the host's defenses might be considered optimistic, but they will at least tell us a great deal about ***tumor*** cell behavior and the ability of the host to influence it. (44 Refs.)

7/7/155 (Item 155 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07884100 92022100
Immunotherapy of breast ***cancer*** : a review of the development of cell-specific therapy.
Lytle GH

Department of Surgery, University of Oklahoma College of Medicine-Tulsa 74129.
Semin Surg Oncol (UNITED STATES) Jul-Aug 1991, 7 (4) p211-6, ISSN 8756-0437 Journal Code: SSO
Languages: ENGLISH
Document type: JOURNAL ARTICLE; ***REVIEW***; REVIEW, TUTORIAL A brief history of immunotherapy for breast ***cancer*** is presented, with emphasis on how theories developed as the field of immunology became more sophisticated. Non-specific therapies, such as Bacillus Calmette-Guerin, levamisole, interferon, interleukin, and others are reviewed. A form of cell-specific immunotherapy is then presented, and some current results are summarized. Problems and proposals for the future development of immunotherapy for breast ***cancer*** are then presented. (69 Refs.)

7/7/148 (Item 148 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07958157 92096157
Cellular and humoral immune responses against ***cancer***: implications for ***cancer*** ***vaccines***.
Knuth A; Wolfel T; Meyer zum Buschenfelde KH
Klinikum, Johannes-Gutenberg-Universitat, Mainz, Germany. Curr Opin Immunol (ENGLAND) Oct 1991, 3 (5) p659-64, ISSN 0952-7915 Journal Code: AH1
Languages: ENGLISH
Document type: JOURNAL ARTICLE; ***REVIEW***; REVIEW, TUTORIAL The key issue in ***tumor*** immunology is to identify antigens as target structures for a ***cancer***-selective immunological attack in the ***tumor***-bearing host, resulting in ***tumor*** rejection. There is a growing detailed understanding of structural and regulatory gene alterations giving rise to candidate rejection antigens and peptides in ***tumor*** cells. As well as reviewing the development of new adjuvant and recombinant vector systems, new approaches are suggested for the construction of ***cancer*** ***vaccines***. (56 Refs.)

7/7/121 (Item 121 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08135156 92273156
Tumor antigens.
Urban JL; Schreiber H
Department of Pathology, University of Chicago, Illinois 60637. Annu Rev Immunol (UNITED STATES) 1992, 10 p617-44, ISSN 0732-0582 Journal Code: ALO
Contract/Grant No.: CA-37156, CA, NCI; CA-22677, CA, NCI; CA-19266, CA, NCI
Languages: ENGLISH
Document type: JOURNAL ARTICLE; ***REVIEW***; REVIEW, ACADEMIC This review solidifies a new concept that common and rare types of human cancers harbor a variety of ***tumor***-specific mutant proteins that may be recognized as ***tumor***-specific antigens. These mutant proteins are encoded by oncogenes or suppressor genes that have undergone structural mutations resulting from point mutations, chromosomal translocations, internal deletions and viral insertional mutagenesis; several of these changes result in fusion proteins. While there is no evidence that immunosurveillance against these mutant proteins can prevent the development of primary cancers without prior immunization of the host, such ***tumor*** -specific molecules might be important for diagnosis and as targets for specific immunotherapy once the ***cancer*** has developed or even as targets for preventive ***cancer*** ***vaccines*** . Evidence further supports the notion that cytolytic or helper T cells are exquisitely selective in recognizing intracellular mutant proteins, and ***tumor*** -specific T cell clones presently

available may become useful for identifying previously unrecognized ***tumor***-specific mutations. Many ***tumor***-specific mutant proteins clearly play a causative role in the establishment of malignant behavior, whereas other carcinogen-induced changes have at least immunological relevance. In any case strong evidence in mouse and man indicates that a single malignant cell can express multiple independent antigenic target sites. Such multiplicity may allow a multi-pronged immune attack that substantially decreases the chance of ***tumor*** escape. Future work must explore whether immune responses to ***tumor***-specific mutant proteins can lead to immunological ***tumor*** rejection and explore the possibility of chemically engineering ***tumor*** mutant peptides to be highly immunogenic, even in hosts that have previously failed to respond to the ***tumor***. (162 Refs.)

7/7/118 (Item 118 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08150607 92288607
Recent advances in antitumor ***vaccines***.
Hu SL; Hellstrom I; Hellstrom KE
Biotechnology (UNITED STATES) 1992, 20 p327-43, ISSN 0740-7378 Journal Code: BIT
Languages: ENGLISH
Document type: JOURNAL ARTICLE; ***REVIEW***; REVIEW, TUTORIAL Immunization with anti-idiotypic antibodies can induce cell-mediated and humoral antitumor immunity in animal models. This immunity can sometimes cause ***tumor*** destruction. However, more needs to be learned about how best to induce the type of immune response that is responsible for ***tumor*** destruction, since the presence of anti-idiotypic antibodies has been shown occasionally to enhance, rather than to inhibit, ***tumor*** growth. There is evidence suggesting that immunization of human ***cancer*** patients with Ab2 can have therapeutic benefit, and also that patients who mount a vigorous Ab2 response following treatment with an Ab1 may do clinically better than those who do not make any Ab2. Although the generation of Ab2 related to infused antitumor Ab1 does not cause ***tumor*** rejection in the majority of patients, and although the clinical data from patients given Ab2 are scarce, the suggestion that Ab2 may cause destruction of human cancers indicates that further work in this area may become rewarding. (83 Refs.)
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E6 1 AU=BERDA P
E7 1 AU=BERDACH G
E8 1 AU=BERDAEV SIU
E9 1 AU=BERDAGUER P
E10 1 AU=BERDAH C
E11 2 AU=BERDAH H
E12 15 AU=BERDAH J

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51 AU=BERD D
188855 CANCER
233518 TUMOR
30510 MELANOMA
S1 39 AU="BERD D" AND (CANCER OR TUMOR OR MELANOMA) ?t s1/6/1-39

1/6/1
08817387 94132387
Expression of platelet-endothelial cell adhesion molecule-1 (PECAM-1) during ***melanoma***-induced angiogenesis in vivo.

1/6/2
08668309 93378309
Treatment of human ***melanoma*** with a hapten-modified autologous vaccine.

1/6/3
08469765 93179765
Autologous ***melanoma*** vaccine induces inflammatory responses in ***melanoma*** metastases: relevance to immunologic regression and immunotherapy.

1/6/4
08027417 92165417
Effective combination chemo/hormonal therapy for malignant ***melanoma*** : experience with three consecutive trials.

1/6/5
07852343 91371343
Growth and metastasis of fresh human ***melanoma*** tissue in mice with severe combined immunodeficiency.

1/6/6
07696616 91215616
Immunization with haptenized, autologous ***tumor*** cells induces inflammation of human ***melanoma*** metastases.

1/6/7
07532780 91051780
A randomized study of methanol-extraction residue of bacille Calmette-Guerin as postsurgical adjuvant therapy of uveal ***melanoma***.

1/6/8
07529360 91048360
Time to recurrence varies inversely with thickness in clinical stage 1 cutaneous ***melanoma***.

1/6/9
07519176 91038176
Treatment of metastatic ***melanoma*** with an autologous ***tumor*** -cell vaccine: clinical and immunologic results in 64 patients.

1/6/10
07151535 90058535
Flow cytometric determination of the frequency and heterogeneity of expression of human ***melanoma***-associated antigens.

1/6/11
07151491 90058491
Depletion of T-cells with the CD4+CD45R+ phenotype in lymphocytes that infiltrate subcutaneous metastases of human ***melanoma***.

1/6/12
07049453 89351453
Immunotherapy of ***cancer*** using IL-2 [letter; comment]

1/6/13
06987200 89289200
Flow cytometric evaluation of bronchoscopic washings and lavage fluid for DNA aneuploidy as an adjunct in the diagnosis of lung ***cancer*** and tumors metastatic to the lung.

1/6/14
06938817 89240817
Low doses of chemotherapy to inhibit suppressor T cells.

1/6/15
06849243 89151243

The importance of tamoxifen to a cisplatin-containing regimen in the treatment of metastatic ***melanoma***.

1/6/16
06782598 89084598
Newer immunologic approaches to the treatment of patients with ***melanoma***.

1/6/17
06700199 89002199
Active immunotherapy of human ***melanoma*** exploiting the immunopotentiating effects of cyclophosphamide.

1/6/18
06505817 88150817
Effect of low dose cyclophosphamide on the immune system of ***cancer*** patients: depletion of CD4+, 2H4+ suppressor-inducer T-cells.

1/6/19
06497189 88142189
Elimination of immune suppressor mechanisms in humans by oxazaphosphorines.

1/6/20
06241639 87215639
Effect of low dose cyclophosphamide on the immune system of ***cancer*** patients: reduction of T-suppressor function without depletion of the CD8+ subset.

1/6/21
06213329 87187329
Combination chemotherapy and hormonal therapy in the treatment of malignant ***melanoma***.

1/6/22
06213145 87187145
Depletion of suppressor-cytotoxic T-lymphocytes by administration of a murine monoclonal antibody.

1/6/23
05934495 86235495
The immunoaugmenting effects of ***cancer*** chemotherapeutic agents.

1/6/24
05888578 86189578
Induction of cell-mediated immunity to autologous ***melanoma*** cells and regression of metastases after treatment with a ***melanoma*** cell vaccine preceded by cyclophosphamide.

1/6/25
05609021 85225021
Metastatic uveal ***melanoma***. Pretherapy serum liver enzyme and liver scan abnormalities.

1/6/26
05408621 85024621
Potentiation of human cell-mediated and humoral immunity by low-dose cyclophosphamide.

1/6/27
05182637 84106637
Current condition and prognosis of ***tumor*** immunotherapy: a second opinion.

1/6/28
05182593 84106593
Impairment of concanavalin A-inducible suppressor activity following administration of cyclophosphamide to patients with advanced ***cancer***.

1/6/29
04970590 83203590
Metastatic uveal ***melanoma*** . Hepatic cell-surface enzymes, isoenzymes, and serum sialic acid levels in early metastatic disease.

1/6/30
04791804 83024804
Augmentation of the human immune response by cyclophosphamide.

1/6/31
03941631 80052631
Evaluation of a "nude" mouse-human ***tumor*** panel as a predictive secondary screen for ***cancer*** chemotherapeutic agents.

1/6/32
03933675 80044675
Effect of 1-(2-chloroethyl)-3-(4-methylcyclohexyl)-1-nitrosourea adjuvant therapy on the immune response of patients with malignant ***melanoma***.

1/6/33
03812578 79189578
Positive phase II trial of dibromodulcitol in patients with metastatic ***melanoma*** refractory to DTIC and a nitrosourea.

1/6/34
03792390 79169390
Chemoimmunotherapy increases the lymphocyte reactivity of ***melanoma*** patients.

1/6/35
03533238 78167238
Phase II trial of VM-26 in patients with metastatic malignant ***melanoma***.

1/6/36
03479552 78113552
Phase II study of subcutaneously administered 5-azacytidine (NSC-102816) in patients with metastatic

malignant ***melanoma***.

1/6/37
03252299 77154299
Current concepts of the biology of human cutaneous malignant ***melanoma***.

1/6/38
03170399 77072399
Review of immunotherapeutic studies on ***cancer*** patients. pp. 155-70.

1/6/39
03162191 77064191
Critical review of previously reported clinical trials of ***cancer*** immunotherapy with nonspecific immunostimulants.
?s e4

S2 2 AU="BERD DA"
?t s2/6/1-2

2/6/1
03121715 77023715
Immunological enhancement of leukemia L1210 by Corynebacterium parvum in allogeneic mice.

2/6/2
03104499 77006499
A study of antitumor (phase II) and immunosuppressive effects of ICRF-159 in patients with metastatic melanoma.
?t s2/7/2

2/7/2
DIALOG(R)File 155:MEDLINE(R)

03104499 77006499
A study of antitumor (phase II) and immunosuppressive effects of ICRF-159 in patients with metastatic melanoma.
Bellet RE; Catalano RB; Danna VG; ***Berd DA***; Berkelhammer J; Mastrangelo MJ
J Clin Pharmacol (UNITED STATES) Aug-Sep 1976, 16 (8-9) p433-8, ISSN 0091-2700 Journal Code: HT9
Languages: ENGLISH
Document type: CLINICAL TRIAL; JOURNAL ARTICLE
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>>> PAUSE ended.

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DIALOG(R)File 155:MEDLINE(R)

08668309 93378309
Treatment of human ***melanoma*** with a hapten-modified autologous vaccine.
Berd D; Maguire HC Jr; Mastrangelo MJ
Department of Medicine, Thomas Jefferson University, Philadelphia, Pennsylvania 19107.
Ann N Y Acad Sci (UNITED STATES) Aug 12 1993, 690 p147-52, ISSN 0077-8923 Journal Code: 5NM
Contract/Grant No.: CA 39248, CA, NCI
Languages: ENGLISH
Document type: CLINICAL TRIAL; JOURNAL ARTICLE

1/7/3
DIALOG(R)File 155:MEDLINE(R)

08469765 93179765
Autologous ***melanoma*** vaccine induces inflammatory responses in ***melanoma*** metastases: relevance to immunologic regression and immunotherapy.
Murphy GF; Radu A; Kaminer M; ***Berd D***
Department of Dermatology, University of Pennsylvania School of Medicine, Philadelphia.
J Invest Dermatol (UNITED STATES) Mar 1993, 100 (3) p335S-341S, ISSN 0022-202X Journal Code: IHZ
Contract/Grant No.: CA-40358, CA, NCI; CA-39248, CA, NCI Languages: ENGLISH
Document type: JOURNAL ARTICLE
Human primary malignant ***melanoma*** is often accompanied by a host response of infiltrating lymphocytes suggestive of ***tumor*** antigen-induced immunity and correlated in some tumors with prognosis. Whereas metastatic ***melanoma*** deposits typically are not inflamed and contain relatively few lymphocytes and dendritic immune cells, immunization with autologous ***melanoma*** -cell vaccine may induce a clinical inflammatory response associated with mononuclear-cell infiltration. In this study, we characterize immune responses to dermal and subcutaneous ***melanoma*** metastases in dinitrophenyl (DNP)-pre-sensitized patients immunized with DNP-conjugated ***melanoma*** cells. Patients so treated develop cutaneous delayed hypersensitivity responses to DNP-conjugated autologous mononuclear cells, and approximately one-half show clinical evidence of inflammation and regression of metastases within 2-4 months. Whereas pre-vaccination biopsies of metastatic ***melanoma*** failed to reveal significant infiltration by lymphocytes, biopsies obtained after vaccination and coincident with clinical inflammation were markedly infiltrated preponderantly by T cells with a CD8+ phenotype. Clustering of these cells about individual degenerating ***melanoma*** cells in a manner analogous to "satellitosis" was a consistent feature of this reaction. Enhanced expression of intercellular adhesion molecule-1 (ICAM-1) and human leukocyte antigen (HLA)-DR by ***melanoma*** cells were invariably associated with zones of T-cell infiltration, whereas diminished or absent expression was observed in relatively unaffected regions of tumors. Numerous HLA-DR+, CD4+, CD1-, Leu-1- dendritic cells were also associated with zones of early T-cell infiltration. These data indicate that clinical inflammation and regression of metastatic ***melanoma*** induced by autologous ***melanoma*** -cell vaccine involves activated T cells with cytotoxic-suppressor phenotype and dendritic cells putatively capable of local antigen presentation. ICAM-1 upregulation on ***melanoma*** cells is a likely mediator of ligand interaction between infiltrating T cells and target cells in this model of antigen-induced host anti-***tumor*** response. Structural alterations identified in this setting (e.g., ***tumor*** cell satellitosis) may provide additional insight into identifying features of naturally occurring host immune responses to primary cutaneous melanomas.

1/7/4

DIALOG(R)File 155:MEDLINE(R)

08027417 92165417
Effective combination chemo/hormonal therapy for malignant ***melanoma*** : experience with three consecutive trials.
McClay EF; Mastrangelo MJ; ***Berd D***; Bellet RE
Cancer Center, University of California, San Diego.
Int J Cancer (UNITED STATES) Feb 20 1992, 50 (4) p553-6, ISSN 0020-7136 Journal Code: GQU
Languages: ENGLISH
Document type: CLINICAL TRIAL; JOURNAL ARTICLE
Our experience with the combination of dacarbazine, carmustine, cisplatin with and without tamoxifen is reported. In our initial study, with all 4 drugs, we had an overall response rate of 50% with a complete response rate of 15%. Due to a high incidence of deep venous thrombosis and the lack of effectiveness of tamoxifen as a single agent, we deleted tamoxifen from the regimen and treated another 20 patients. Surprisingly, the response rate decreased to 10%. We then re-incorporated tamoxifen into the regimen and treated 25 additional patients. In this third group of patients we experienced an objective response rate of 52% with a complete response rate of 8%. Overall, 65 patients have been treated: 45 with and 20 without tamoxifen. Twenty-three (51%) patients treated with tamoxifen have responded, with 5 (11%) patients achieving a complete response. Only 2 (10%) patients treated without tamoxifen have responded. Despite the improvement in the response rate, a corresponding increase in survival has not been seen. Patients treated with tamoxifen had a mean survival of 10.8 (SD 13.6) months compared with a mean survival of 9.8 (SD 7.3) months for those treated without tamoxifen. The absence of survival advantage for the tamoxifen-treated patients may be due to early failure in the central nervous system. In 48% of the responding tamoxifen-treated patients, the first site of failure was the central nervous system, while systemic disease was still responding.

1/7/6
DIALOG(R)File 155:MEDLINE(R)

07696616 91215616
Immunization with haptenized, autologous ***tumor*** cells induces inflammation of human ***melanoma*** metastases.
Berd D; Murphy G; Maguire HC Jr; Mastrangelo MJ
Thomas Jefferson University, Division of Medical Oncology, Philadelphia, Pennsylvania 19107.
Cancer Res (UNITED STATES) May 15 1991, 51 (10) p2731-4, ISSN 0008-5472 Journal Code: CNF
Contract/Grant No.: CA 39248, CA, NCI; CA 40358, CA, NCI; AR 39674, AR, NIAMS
Languages: ENGLISH
Document type: JOURNAL ARTICLE
Twenty-four patients with metastatic ***melanoma*** were treated with a novel form of active immunotherapy, autologous ***tumor*** cell vaccine conjugated to the hapten, dinitrophenyl. This approach is based on the idea, well established in animal systems, that presentation of ***tumor*** antigens in the context of a strongly immunogenic hapten augments the development of immunity to those antigens. After being sensitized to dinitrophenyl, patients were given injections of dinitrophenyl-vaccine every 28 days following pretreatment with low dose cyclophosphamide. The vaccine induced a striking inflammatory response in superficial metastases in 14 of 24 patients, consisting of erythema, swelling, warmth, and tenderness over ***tumor*** masses. Immunohistochemistry and flow cytometric analysis of biopsy specimens showed marked infiltration with lymphocytes, the majority of which were CD8+, HLA-DR+ T-cells. These observations suggest that a T-cell-mediated immune response against ***melanoma*** -associated antigens was facilitated by the "helper" effect of the anti-hapten response.

1/7/7
DIALOG(R)File 155:MEDLINE(R)

07532780 91051780
A randomized study of methanol-extraction residue of bacille Calmette-Guerin as postsurgical adjuvant therapy of uveal ***melanoma***. McLean IW; ***Berd D***; Mastrangelo MJ; Shields JA; Davidorf FH; Grever M; Makley TA; Gamel JW
Department of Ophthalmic Pathology, Armed Forces Institute of Pathology, Washington, D.C. 20306-6000.
Am J Ophthalmol (UNITED STATES) Nov 15 1990, 110 (5) p522-6, ISSN 0002-9394 Journal Code: 3OQ
Contract/Grant No.: EY04482, EY, NEI; CA40526, CA, NCI; CA39248, CA, NCI Languages: ENGLISH
Document type: CLINICAL TRIAL; JOURNAL ARTICLE; RANDOMIZED CONTROLLED TRIAL
A randomized controlled clinical trial of methanol-extracted residue of bacille Calmette-Guerin adjuvant treatment of posterior uveal ***melanoma*** was undertaken. Of 113 patients, 34 patients received adjuvant immunotherapy and 79 patients received no treatment. No difference in survival was observed between the adjuvant-treated group and the control group of patients. This study found that the size of the ***tumor*** was a highly significant risk factor for death caused by metastasis of uveal melanomas. The standard deviation of the nucleolar area of the neoplastic cells was a significant risk factor, even though patients with tumors composed of Callender's spindle-type cells were not included in the study.

1/7/8
DIALOG(R)File 155:MEDLINE(R)

07529360 91048360
Time to recurrence varies inversely with thickness in clinical stage 1 cutaneous ***melanoma***.
Schultz S; Kane M; Roush R; Miller V; ***Berd D***; Goldman L; Mastrangelo M
Memorial Clinic of Indianapolis, Indiana.
Surg Gynecol Obstet (UNITED STATES) Nov 1990, 171 (5) p393-7, ISSN 0039-6087 Journal Code: VBD
Languages: ENGLISH
Document type: JOURNAL ARTICLE
The thickness of a ***tumor*** has been identified as the principal prognostic factor in cutaneous malignant ***melanoma***. However, time to recurrence has not conclusively been related to thickness. A retrospective study of 216 patients with a primary cutaneous malignant ***melanoma*** that recurred was conducted to clarify this relationship and investigate possible independent relationships between age at diagnosis and sex of patients to time to recurrence. The results of analysis of linear regression revealed an inverse linear relationship between thickness and time to recurrence (p less than 0.001). Patients more than 50 years of age at the time of diagnosis were shown to relapse sooner than those less than 50 years of age ($p = 0.014$). Sex was not a significant factor in predicting time to recurrence (p greater than 0.10). These results suggest that thickness of ***tumor*** provides an indication of time to recurrence in those patients destined to recur and stress the need for long term surveillance in patients with a history of malignant ***melanoma*** because of the possibility of late relapse even with thin lesions.

1/7/9
DIALOG(R)File 155:MEDLINE(R)

07519176 91038176
Treatment of metastatic ***melanoma*** with an autologous ***tumor*** -cell vaccine: clinical and immunologic results in 64 patients. ***Berd D***; Maguire HC Jr; McCue P; Mastrangelo MJ
Department of Medicine, Thomas Jefferson University, Philadelphia, PA 19107.

J Clin Oncol (UNITED STATES) Nov 1990, 8 (11) p1858-67, ISSN 0732-183X Journal Code: JCO
Contract/Grant No.: CA 39248, CA, NCI
Languages: ENGLISH
Document type: JOURNAL ARTICLE

We treated 64 patients with metastatic ***melanoma*** using a ***melanoma*** vaccine preceded by low-dose cyclophosphamide (CY), and monitored immunologic effects and antitumor activity. On day 0, the patients were given CY 300 mg/m2 intravenously. Three days later, they were injected intradermally with vaccine consisting of 10 to 25 x 10(6) autologous, enzymatically dissociated, cryopreserved, irradiated (25 Gy) ***tumor*** cells mixed with bacillus Calmette-Guerin (BCG). This treatment sequence was repeated every 28 days. Of 40 assessable patients with measurable metastases, five had responses, four complete and one partial, with a median duration of 10 months (7 to 84+ months). In six additional patients, we observed an antitumor response that seems to be peculiar to this vaccine therapy: the regression of metastatic lesions that appeared after the immunotherapy was begun. Delayed-type hypersensitivity (DTH) to autologous, mechanically dissociated ***melanoma*** cells that had not been exposed to extraneous antigens, such as enzymes or fetal calf serum, increased significantly following immunotherapy (day 0 v day 49, P less than .001; day 0 v day 161, P less than .001; day 0 v day 217, P = .021). Antitumor responses to the vaccine were strongly associated with DTH, as indicated by three observations: (1) eight of 10 patients who exhibited ***tumor*** regression had positive DTH, (2) in postsurgical adjuvant patients, there was a highly significant linear relationship (P less than .001) between the intensity of DTH to autologous ***melanoma*** cells and the time to recurrence of ***tumor***, and (3) nine patients who developed DTH to the autologous ***melanoma*** cells in their original vaccine developed new metastases that failed to elicit DTH or elicited a much smaller response. In three cases, we were able to excise regressing tumors for histologic examination; such tumors were characterized by an intense infiltration of lymphocytes. This demonstration that an immune response to ***melanoma***-associated antigens can be elicited in ***cancer***-bearing patients provides some basis for optimism about the prospects for developing active immunotherapy that has practical therapeutic value.

1/7/16
DIALOG(R)File 155:MEDLINE(R)

06782598 89084598
Newer immunologic approaches to the treatment of patients with ***melanoma***.
Mastrangelo MJ; Schultz S; Kane M; ***Berd D***
Division of Medical Oncology, Jefferson Medical College, Philadelphia, PA.
Semin Oncol (UNITED STATES) Dec 1988, 15 (6) p589-94, ISSN 0093-7754 Journal Code: UN5
Languages: ENGLISH
Document type: JOURNAL ARTICLE; REVIEW; REVIEW, ACADEMIC (54 Refs.)

1/7/17
DIALOG(R)File 155:MEDLINE(R)

06700199 89002199
Active immunotherapy of human ***melanoma*** exploiting the immunopotentiating effects of cyclophosphamide.
Berd D; Mastrangelo MJ
Division of Medical Oncology, Thomas Jefferson University, Philadelphia, Pennsylvania 19107.
Cancer Invest (UNITED STATES) 1988, 6 (3) p337-49, ISSN 0735-7907 Journal Code: CAI
Contract/Grant No.: CA39248
Languages: ENGLISH
Document type: JOURNAL ARTICLE

Malignant tumors may escape rejection by the immune system because they induce a state of immunological tolerance mediated by ***tumor*** antigen-specific suppressor T cells. In animal systems, cyclophosphamide can reverse the tolerance and thereby facilitate immunologically mediated ***tumor*** destruction. We have applied these concepts to the immunotherapy of human malignant ***melanoma***. Forty-three patients with metastatic disease were treated with a whole cell vaccine 3 days after intravenous administration of cyclophosphamide, 300 mg/m2. The vaccine consisted of cryopreserved, irradiated autologous ***melanoma*** cells, obtained from metastatic masses by dissociation with collagenase and DNAse, mixed with bacillus Calmette-Guerin (BCG) and injected intradermally. The cyclophosphamide (CY) + vaccine combination was repeated every 28 days. Delayed-type hypersensitivity (DTH) was tested by injecting 1 x 10(6) ***melanoma*** cells intradermally and measuring the diameter of induration at 48 h. Most patients had minimal pretreatment DTH responses to ***melanoma*** cells (mean +/- SE, mm = 2.4 +/- 0.5). After two vaccine treatments, the responses increased significantly (mean increase +/- SE = 12.1 +/- 1.6 p less than .001) and that level of response was maintained after 4, 6, and 8 treatments. The patients were also skin-tested with a mixture of the enzymes used to dissociate the tumors. No patients exhibited DTH to collagenase + DNAse prior to vaccine injection, but every patient developed DTH to the mixture following two treatments (mean, mm = 26.4 +/- 3.9). Although extracting viable cells from ***tumor*** tissue without the use of enzymes proved difficult, we were able to test DTH to mechanically dissociated ***tumor*** cells in 23 patients. After two vaccine treatments, there was a significant increase in DTH to enzyme-free autologous ***melanoma*** cells (mean DTH +/- SE, mm: 5.4 +/- 1.0, p less than .01). Whereas 5 of 23 patients had positive DTH responses (5 mm induration or greater) before treatment, 11 of 23 were positive after two treatments. Further significant increases in DTH enzyme-free cells were observed after 6 and 8 treatments. Thus, it appears that patients receiving CY + vaccine developed DTH to ***tumor*** -associated antigens as well as to residual collagenase and DNAse on the cell surface. Thirty-three patients could be evaluated for antitumor effects of cyclophosphamide + vaccine. There were 3 complete remissions, 1 partial remission, and 2 minor responses. Two complete responders remain alive and free of disease after 57 and 12 months, respectively, and the third died after 39 months. The partial remission consisted of 75% regression of a pulmonary metastasis.(ABSTRACT TRUNCATED AT 400 WORDS)

1/7/18
DIALOG(R)File 155:MEDLINE(R)

06505817 88150817
Effect of low dose cyclophosphamide on the immune system of ***cancer*** patients: depletion of CD4+, 2H4+ suppressor-inducer T-cells. ***Berd D***; Mastrangelo MJ
Department of Medicine, Thomas Jefferson University, Philadelphia, Pennsylvania 19107.
Cancer Res (UNITED STATES) Mar 15 1988, 48 (6) p1671-5, ISSN 0008-5472 Journal Code: CNF
Contract/Grant No.: CA39248
Languages: ENGLISH
Document type: JOURNAL ARTICLE
We studied peripheral blood lymphocytes (PBL) from 42 patients with metastatic ***melanoma*** undergoing treatment with cyclophosphamide (CY) plus ***melanoma*** vaccine to determine whether CY immunopotentiation could be related to depletion of T-cells that function as inducers of suppression. Every 28 days, the patients were given CY, 300 mg/m2 i.v., followed 3 days later by the intradermal injection of autologous, irradiated ***melanoma*** cells mixed with Bacillus Calmette-Guerin. PBL were separated by density gradient centrifugation and cryopreserved until needed for testing. They were stained with monoclonal antibodies directly conjugated to fluorescein isothiocyanate or phycoerythrin and analyzed by two-color flow cytometry. At no time after the initiation of CY plus vaccine were there any significant changes in the percentages of helper-inducer T-cells (CD4+), suppressor-cytotoxic T-cells (CD8+), or the subpopulation of CD8+ cells expressing Leu 15, a marker for suppressor cells. Treatment of ***melanoma*** patients with CY plus vaccine resulted in a progressive fall in the proportion of CD4+

T-cells expressing the 2H4 (CD45) antigen, which identifies inducers of suppression. The reduction of CD4+, 2H4+ T-cells did not become apparent until day 28 after the first dose of CY and reached statistical significance only on days 49 (21 days after the second dose) and 105 (21 days after the fourth dose) (mean changes +/- SE: day 49, -5.4 +/- 1.4%, P less than 0.01; day 105, -9.1 +/- 2.2%, P less than 0.01; t test for nonindependent samples). In contrast, the proportion of CD4+ T-cells expressing the antigen 4B4 (CDw29), which are true helper cells, increased slightly, although not significantly, following the institution of CY plus vaccine (mean changes: day 49, +2.9 +/- 2.1%; day 105, +3.6 +/- 2.4%). Similar results were obtained when absolute numbers of circulating cells, rather than percentages, were analyzed. Thus the number of CD4+, 2H4+ T-cells fell from a mean of 395,000/ml on day 0 to 309,000/ml on day 49 (P less than 0.01) to 256,000/ml on day 105 (P less than 0.05). The absolute number of CD4+, 4B4+ cells remained unchanged at the same time points. These changes were not due to progression of metastatic disease, since a comparison of patients with progressive metastases with those who were rendered disease free by surgery showed no significant differences in the reduction of the percentage of CD4+, 2H4+ T-cells.(ABSTRACT TRUNCATED AT 400 WORDS)

1/7/21
DIALOG(R)File 155:MEDLINE(R)

06213329 87187329
Combination chemotherapy and hormonal therapy in the treatment of malignant ***melanoma***.
McClay EF; Mastrangelo MJ; Bellet RE; ***Berd D***
Cancer Treat Rep (UNITED STATES) May 1987, 71 (5) p465-9, ISSN 0361-5960 Journal Code: CNM
Languages: ENGLISH
Document type: JOURNAL ARTICLE
Twenty-three patients with metastatic ***melanoma*** were treated with combination therapy consisting of dacarbazine (220 mg/m2) and cisplatin (25 mg/m2) iv daily for 3 days every 3 weeks, carmustine (150 mg/m2) iv every 6 weeks, and tamoxifen (10 mg) orally twice daily. In 20 evaluable patients, there were no complete responses and ten partial responses. The median remission duration has not yet been reached but exceeds 7 months. Treatment was relatively well tolerated. However, six patients developed deep venous thrombosis, and four of these six suffered pulmonary emboli. Our data support a previous study and suggest that this combination warrants comparison with the active single components in a randomized prospective trial.

1/7/24
DIALOG(R)File 155:MEDLINE(R)

05888578 86189578
Induction of cell-mediated immunity to autologous ***melanoma*** cells and regression of metastases after treatment with a ***melanoma*** cell vaccine preceded by cyclophosphamide.
Berd D; Maguire HC Jr; Mastrangelo MJ
Cancer Res (UNITED STATES) May 1986, 46 (5) p2572-7, ISSN 0008-5472 Journal Code: CNF
Contract/Grant No.: CA39248
Languages: ENGLISH
Document type: JOURNAL ARTICLE
There is considerable evidence in animal ***tumor*** systems that antitumor immunity is modulated by suppressor T-lymphocytes, and that the cytotoxic drug cyclophosphamide (CY) can abrogate that suppression. We measured the acquisition of delayed-type hypersensitivity (DTH) to autologous ***melanoma*** cells in 19 patients with metastatic malignant ***melanoma***. The patients were treated with an autologous ***melanoma*** cell vaccine, either given alone, or given 3 days after the administration of CY, 300 mg/m2 i.v. The DTH responses of CY-pretreated patients were significantly greater than those of control (vaccine only) patients. Thus, after two vaccine treatments, the median DTH responses (mm

induration) were as follows: controls, 4 mm; CY pretreated, 11 mm; P = 0.034, Mann-Whitney U test, 2-tailed. Whereas seven of eight CY-pretreated patients developed DTH to autologous ***melanoma*** cells of at least 5 mm, only two of seven controls did so (P = 0.034, Fisher's exact test). Two patients had significant antitumor responses to treatment with CY plus vaccine, consisting of complete disappearance of skin metastases and a pulmonary nodule in one, and regression of s.c. and liver metastases in the other. Both patients remain free of ***melanoma*** after 42 and 33 mo, respectively.

1/7/26
DIALOG(R)File 155:MEDLINE(R)

05408621 85024621
Potentiation of human cell-mediated and humoral immunity by low-dose cyclophosphamide.
Berd D; Maguire HC Jr; Mastrangelo MJ
Cancer Res (UNITED STATES) Nov 1984, 44 (11) p5439-43, ISSN 0008-5472 Journal Code: CNF
Contract/Grant No.: CA32123
Languages: ENGLISH
Document type: JOURNAL ARTICLE
Although cyclophosphamide (CY) is a potent immunosuppressive drug, under the proper conditions, it can potentiate immune responses as well. In past work, we have shown that administration of a commonly used oncostatic dose of CY (1000 mg/sq m) to patients with advanced ***cancer*** 3 days before sensitization with the primary antigen, keyhole limpet hemocyanin (KLH), resulted in augmentation of delayed-type hypersensitivity (DTH) but not antibody response to that antigen. The present study was performed to test the immunopotentiation of a lower dose of CY (300 mg/sq m); animal studies and studies of human lymphocytes in vitro suggested that the lower dose might be more effective. Eighteen patients with advanced metastatic ***cancer*** were alternately assigned to one of two groups. Sixteen days before CY, one group received KLH and the other group received 1-chloro-2,4-dinitrobenzene (DNCB). CY 300 mg/sq m was given as an i.v. bolus on Day 0. Three days after CY, the patients received KLH or DNCB, whichever they had not received initially. Blood was drawn for antibody titer, and skin testing was performed 14 days after administration of KLH or DNCB. In addition, skin tests to microbial recall antigens were made 2 days before and 17 days after CY. Pretreatment with low-dose CY resulted in significant augmentation of DTH to KLH; thus, the median DTH responses were: KLH alone, 10 mm; and KLH after CY, 27 mm (p less than 0.01). CY pretreatment also resulted in augmentation of the antibody response to KLH. The median total antibody titers (log2 of reciprocal of dilution) were as follows: KLH alone, less than 1; and KLH after CY, 3 (p less than 0.01). All nine CY-pretreated subjects but only 4 of 9 controls developed measurable anti-KLH antibody titers. CY pretreatment neither augmented nor suppressed the 48-hr challenge reaction to DNCB. Moreover, CY had no effect on DTH responses to the recall antigens, dermatophytin, Candida, and mumps.

1/7/30
DIALOG(R)File 155:MEDLINE(R)

04791804 83024804
Augmentation of the human immune response by cyclophosphamide. ***Berd D***; Mastrangelo MJ; Engstrom PF; Paul A; Maguire H Cancer Res (UNITED STATES) Nov 1982, 42 (11) p4862-6, ISSN 0008-5472 Journal Code: CNF
Contract/Grant No.: CA-13456
Languages: ENGLISH
Document type: JOURNAL ARTICLE
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009794450 WPI Acc No: 94-074303/09
XRAM Acc No: C94-033785
Tumour vaccine contains melanoma cell conjugated to hapten, eg. DNP - useful for treating melanoma

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009794450 WPI Acc No: 94-074303/09
XRAM Acc No: C94-033785
Tumour vaccine contains melanoma cell conjugated to hapten, eg. DNP - useful for treating melanoma
Patent Assignee: (UYJE-) UNIV JEFFERSON THOMAS
Author (Inventor): ***BERD D***
Number of Patents: 001
Number of Countries: 001
Patent Family:

CC Number	Kind	Date	Week	
US 5290551	A	940301	9409	(Basic)

Priority Data (CC No Date): US 520649 (900508); US 985334 (921204) Abstract (Basic): US 5290551 A
A vaccine useful for treating melanoma comprises irradiated autologous melanoma cells conjugated to

a heptan selected from dinitrophenyl, trinitrophenyl and N-iodoacetyl-N'-5 sulphonic 1-naphthyl ethylene diamine, and mixed with an immunological adjuvant e.g. Bacille Calmette-Guerin (BCG).

USE - It is given intradermally and comprises 10-25 x 10-6 live, pref. DNA-conjugated tumour cells suspended in 0.2 ml Hanks solution with BCG added (0.1 ml).

In an example, sixty four patients were treated with metastatic melanoma using the melanoma vaccine preceded by low close cyclophosphamide(CY). They were monitored for immunological effects and anti-tumour activity. On day O they were given CY (300 mg/m2 i.v.) 3 days later they were given intradermal injections of 10-25 x 10power-6 autologous, cryopreserved, irradiated (2500R) tumour cells mixed with BCG. The treatment sequence was continued every 28 days. There was 40 evaluable patients with measurable metastases 5 had responses 4 complete, 1 partial. The median division of response was 10 month. Treatment of cancer patient with a haptenised tumour vaccine, preceded by low dose cyclo-phosphamids (CY) induces delayed-type hypersensitivity (DTH) to melanoma cells, and in some cases, regression of metastatic tumours. The efficiency of the process has been increased by immunising with tumour cells conjugated to the hapten such as DNP, TNP or AED. Dwg.0/0

Derwent Class: B04; D16;
Int Pat Class: A61K-037/66; A61K-039/00
?s irradiat? and tumor

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        47387  IRRADIAT?
          380  TUMOR
    S2     21  IRRADIAT? AND TUMOR
?t s2/6/1-21
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2/6/1
008709865 WPI Acc No: 91-213886/29
XRPX Acc No: N91-163117
Radiotherapy in cancer - by ***irradiation*** of primary tumour for 7 and regional metastasis zone for 4 days, repeated 3 weeks later for 3 weeks

2/6/2
008705791 WPI Acc No: 91-209812/29
XRPX Acc No: N91-160173
Optical diffuser for photo-dynamic therapy of oesophageal tumours - supplies visible and infrared radiation via wavelength-independent refractory medium with axially modulated concn. of scatters

2/6/3
007992337 WPI Acc No: 89-257449/36
Related WPI Accession(s): 91-015708
XRAM Acc No: C89-114449
Tumour-specific vaccines - produced by incubating ***irradiated*** tumour cells with Newcastle disease virus

2/6/4
007736143 WPI Acc No: 89-001255/01
XRAM Acc No: C90-131869
Novel vitamin-D-3 derivs. - induce differentiation of tumour cells, and are useful as antitumour agents

2/6/5
007705710 WPI Acc No: 88-339642/48
XRPX Acc No: N88-257535

Microwave therapeutic equipment for treating tumours - has generator arranged to ***irradiate*** patient on couch with microwaves to produce given temperature in lesions

2/6/6
007667888 WPI Acc No: 88-301820/43
XRAM Acc No: C88-133643
Prevention and treatment of skin radiation burning lesions - using novel prepn. contg. 2,6-dimethyl -3,5-diethoxy carbonyl- 1,4-dihydropyridine applied as ointment

2/6/7
007602725 WPI Acc No: 88-236657/34
XRAM Acc No: C88-105835
Treating hypoxic mammalian tumour cells - by administering an oxygenated fluorochemical emulsion interstitially directly to the tumour and ***irradiating***

2/6/8
007311491 WPI Acc No: 87-308498/44
XRAM Acc No: C87-131370
Selectively rendering tissues radio-sensitive - by administration of a mixt. of oxygen and nitrous oxide

2/6/9
007281486 WPI Acc No: 87-278493/40
XRAM Acc No: C87-118330
Dry sepn. of indium from silver matrix - esp. ***irradiated*** target, giving radionuclide useful in nuclear medicine

2/6/10
004739944 WPI Acc No: 86-243286/37
XRAM Acc No: C86-104762
XRPX Acc No: N86-181798
Raw drug contg. organism tissue culture by culturing growth cells e.g. from bud in nutrient contg. medium

2/6/11
004533790 WPI Acc No: 86-037134/06
XRPX Acc No: N86-027075
Ultrasound hyperthermia appts. for treatment of tumours has position detector that detects positional relationship of tomographic ultrasound probe and heating applicator w.r.t. living organism; CANCER

2/6/12
004488321 WPI Acc No: 85-315199/50
XRPX Acc No: N85-234079
Dermal ionising radiation injury prophylaxis and treatment by ultraviolet ***irradiation*** of intact skin section

2/6/13
004464030 WPI Acc No: 85-290908/47

Related WPI Accession(s): 84-101157
XRAM Acc No: C85-125958
XRPX Acc No: N85-216891
Compsn. for sensitising tumours towards destruction by light - contg. high mol. wt. aggregates of dimeric porphyrin cpds.

2/6/14
004166781 WPI Acc No: 84-312320/50
XRAM Acc No: C84-133262
XRPX Acc No: N84-232942
Localisation of cancerous tumours by monitoring IR fluorescence where specific marker present esp. porphyrin

2/6/15
003955613 WPI Acc No: 84-101157/16
Related WPI Accession(s): 85-290908
XRAM Acc No: C84-043126
Haematoporphyrin deriv. for localising and destroying tumours - by ***irradiating*** photosensitised tumour with red light

2/6/16
003918193 WPI Acc No: 84-063737/11
XRPX Acc No: N84-048243
Radiation of hollow organ, e.g. bladder using catheter with light conductor introducing scatter medium locating photosensitised tumour

2/6/17
003700720 WPI Acc No: 83-60702K/25
XRAM Acc No: C83-058923
XRPX Acc No: N83-108508
Diagnosis of liver disease by ***irradiating*** blood serum with ultraviolet light and recording luminescence wavelength; ULTRAVIOLET

2/6/18
003524271 WPI Acc No: 82-72257E/34
Related WPI Accession(s): 87-170154
XRAM Acc No: C82-E72257
Cancer detection with tumour-specific antigens encapsulated in liposome(s) to increase antigenicity

2/6/19
003476455 WPI Acc No: 82-24420E/13
XRAM Acc No: C82-E24420
Enhancing antitumour treatment, e.g. ***irradiation*** or chemotherapy by admin. of 3'-deoxy-guanosine-5'-monophosphate or
3'-deoxy-adenosine-5'-monophosphate

2/6/20
003461477 WPI Acc No: 82-10518J/51
XRAM Acc No: C82-J10518
Chlorophyll deriv. contg. carcinostatic drug having selective activity against tumour cells when

irradiated with visible light; ANTITUMOUR

2/6/21
003459642 WPI Acc No: 82-09600J/51
XRAM Acc No: C82-J09600
Inactivated target cells esp. virus-infected cells useful for stimulating lymphocyte proliferation and assays of cellular immunity ?s irradiat? and vaccine?

47387 IRRADIAT?
4438 VACCINE?
S3 51 IRRADIAT? AND VACCINE?
?s s3 and (tumor or cancer or melanoma

> > > Unmatched parentheses
?s s3 and (tumor or cancer or melanoma)

51 S3
380 TUMOR
7605 CANCER
752 MELANOMA
S4 6 S3 AND (TUMOR OR CANCER OR MELANOMA)
?t s4/6/1-6

4/6/1
009794450 WPI Acc No: 94-074303/09
XRAM Acc No: C94-033785
Tumour ***vaccine*** contains ***melanoma*** cell conjugated to hapten, eg. DNP - useful for treating ***melanoma***

4/6/2
009496483 WPI Acc No: 93-190019/24
XRAM Acc No: C93-084095
Transformed human B-cell line for monoclonal antibody prodn. for ***cancer*** diagnosis - prepd from peripheral blood B-cells of ***cancer*** patients actively immunised with autologous tumour antigen, for treating cancers, isolating and sequencing regions

4/6/3
007992337 WPI Acc No: 89-257449/36
Related WPI Accession(s): 91-015708
XRAM Acc No: C89-114449
Tumour-specific ***vaccines*** - produced by incubating ***irradiated*** tumour cells with Newcastle disease virus

4/6/4
007970935 WPI Acc No: 89-236047/33
XRAM Acc No: C89-105060
Treating ***cancer*** - by inducing photosynthesis in the blood following admin. of chlorophyll, esp. as herbal extract, and ***irradiation***

4/6/5
004098646 WPI Acc No: 84-244187/40
XRAM Acc No: C84-103153
XRPX Acc No: N84-182605
Treatment of ***cancer*** by cell programming with laser light or vaccination with modified ***cancer*** toxin

4/6/6
003459642 WPI Acc No: 82-09600J/51
XRAM Acc No: C82-J09600
Inactivated target cells esp. virus-infected cells useful for stimulating lymphocyte proliferation and assays of cellular immunity ?t s4/7/2,5,7

4/7/2
DIALOG(R)File 351:DERWENT WPI

009496483 WPI Acc No: 93-190019/24
XRAM Acc No: C93-084095
Transformed human B-cell line for monoclonal antibody prodn. for ***cancer*** diagnosis - prepd from peripheral blood B-cells of ***cancer*** patients actively immunised with autologous tumour antigen, for treating cancers, isolating and sequencing regions Patent Assignee: (ALKU) AKZO NV
Author (Inventor): CRICHTON V Z; HASPEL M V; KOBRIN B J
Number of Patents: 009
Number of Countries: 025
Patent Family:

CC Number	Kind	Date	Week	
EP 546634	A2	930616	9324	(Basic)
AU 9230088	A	930617	9331	
NO 9204803	A	930614	9332	
FI 9205638	A	930614	9334	
CA 2083542	A	930614	9336	
ZA 9208880	A	931027	9348	
JP 5317042	A	931203	9402	
HU T65480	T	940628	9429	
AU 651261	B	940714	9432	

Priority Data (CC No Date): US 807300 (911213)
Applications (CC,No,Date): AU 9230088 (921211); EP 92203827 (921209); AU 9230088 (921211); NO 924803 (921211); FI 925638 (921211); CA 2083542 (921123); ZA 928880 (921117); JP 92331961 (921211); HU 923932 (921211) Language: English
EP and/or WO Cited Patents: No-SR.Pub
Designated States
(Regional): AT; BE; CH; DE; DK; ES; FR; GB; GR; IE; IT; LI; LU; MC; NL; PT ; SE
Filing Details: AU0651261 Previous Publ. AU 9230088
Abstract (Basic): EP 546634 A

Transformed human B-cell line 88BV59, ATCC CRL 10624, is new. Pref. a monoclonal antibody (Ab) is produced by 88BV59; and an epitope is reactive with the antibody.

Pref. an aminoacid (AA) sequence comprises the Vh or Vx chain variable region, AAs 1-113 or 1-108, resp., and complementarily determining regions (CDR) specified sub units; and CDRs 1, 2 and 3 or specific Vh and Vx chains.

USE - Useful in the diagnosis and treatment of human cancers. In an example, patients

undergoining surgical resection of colon or rectal cancers were selected for a randomised trial of active specific immuno therapy. Patients had no previous history of ***cancer***, no prior radiation or chemotherapy and in the patient: the tumour extended through the bowel wall (stage B2); had positive lymph nodes (stages C1, C2); or had metastatic disease (stage D). A bowel section was taken, and solid tumour and colon mucosa was sepd.. Patients selected for treatment received 3-weekly intradermal ***vaccine*** injections of 10 power 7 ***irradiated*** autologous tumour cells and 10 power 7 BCG in the first two ***vaccines***, with 10 power 7 tumour cells alone in the final one. Patients were bled at the time of the second injection, 1 week after the first, and at the third vaccination.

Peripheral blood B-cells from immunised patients were exposed to transforming agents to obtain continuously growing cell lines producing monoclonal Abs. Epstein Barr virus was used as the agent, and Ab-producing cells were grown in RPMI 1640 medium + 10% foetal bovine serum, 3 mM L-Glu and 5 microg gentamycin/ml.

Most of the Abs had reduced binding to normal colonic mucosa. Dwg.0/3
Derwent Class: B04; D16;
Int Pat Class: A61K-039/395; C07K-007/06; C07K-007/08; C07K-013/00; C07K-015/06; C12N-005/10; C12N-005/24; C12N-015/07; C12N-015/13; C12P-021/08

4/7/5
DIALOG(R)File 351:DERWENT WPI

004098646 WPI Acc No: 84-244187/40
XRAM Acc No: C84-103153
XRPX Acc No: N84-182605
Treatment of ***cancer*** by cell programming with laser light or vaccination with modified ***cancer*** toxin
Patent Assignee: (HANS/) HANSCHMANN H
Author (Inventor): HANSCHMANN H
Number of Patents: 001
Patent Family:

CC Number	Kind	Date	Week	
DE 3235726	A	840927	8440	(Basic)

Priority Data (CC No Date): DE 3235726 (820927)
Abstract (Basic): DE 3235726

Two methods for treating ***cancer*** by 'programming' ***cancer*** cells. The first comprises subjecting the cells to a shock, specifically laser light, so as to induce release of restriction endonucleases which destroy and defective chromosomes. The second method is to introduce one or more DNA chains, related to the virus or virus-like DNA causing the ***cancer***. This will result in prodn. of an antitoxin against the ***cancer*** toxin.

ADVANTAGE - The method eliminates the need for surgery, high-energy ***irradiation*** and toxic chemotherapeutic agents. @(22pp Dwg.No.0/2)@
Derwent Class: B04; D16; S05;
Int Pat Class: C12Q-001/00

4/7/7
>>>Item 7 is not within valid item range
?t s4/7/6

4/7/6
DIALOG(R)File 351:DERWENT WPI

003459642 WPI Acc No: 82-09600J/51
XRAM Acc No: C82-J09600

Inactivated target cells esp. virus-infected cells useful for stimulating lymphocyte proliferation and assays of cellular immunity Patent Assignee: (KRON/) KRONENBERG L H; (LEEB-) LEE BIOMOLECULAR RE Author (Inventor): KRONENBERG L

Number of Patents: 006

Patent Family:

CC Number	Kind	Date	Week	
EP 66886	A	821215	8251	(Basic)
US 4568542	A	860204	8608	
EP 66886	B	860226	8609	
CA 1200485	A	860211	8611	
DE 3269343	G	860403	8615	
US 4595653	A	860617	8627	

Priority Data (CC No Date): US 269557 (810609); US 709062 (850306) Applications (CC,No,Date): EP 82105034 (820608)

Language: English

EP and/or WO Cited Patents: No.SR.Pub; 8.Jnl.REF; 5.Jnl.REF Designated States (Regional): BE; CH; DE; FR; GB; IT; LI; NL

Filing Details: US4595653 Div.ex 4568542 (945NS)

Abstract (Basic): A) Immunologically reactive particles comprising inactivated target cells are new. (B) Prodn. of the reactive particles involves treatment of the target cells with a psoralen (I) or its deriv. and with long wavelength u.v. radiation.

The virus-infected cells may be influenza virus or Herpes simplex virus infected. The normal cells are esp. nerve cells, muscle cells or tumour tissue. The inactivated target cells may be radiolabelled. Target cells include normal and virus-infected cells, and in the inactivated cells nucleic acids are inactivated but they remain antigenic; and virus-infected cells are no longer infective. The inactivated cells can stimulate virus-specific lymphocyte proliferation and can be used in ***vaccines***; they may also be used for in vitro assays of cellular immunity. The use of (I) and u.v. radiation for inactivation eliminates cell infectivity in a shorter time, and with ***irradiation*** only small amts. of radiant energy are required; recovery of all the cells initially present is possible and native antigenicity is preserved. (30pp)

Abstract (US): 8627 US 4595653

Assay of cellular immunity comprises incubating lymphocytes with inactivated non-infective virus contg. normal cells or tumour cells having cell associated antigens capable of eliciting an immunological response and having had DNA or RNA contained therein covalently bonded to psoralen (deriv.) in the presence of longwavelength UV light. The amt. of lymphocyte proliferation is then measured.

The virus may be Herpes simplex or influenza virus and the cells may be rabbit kidney or Mandin Darby canine cells. @(10pp)@ 8608 US 4568542

Immunologically reactive ***vaccine*** compsn. comprises a medium contg. inactivated, non-infective virus-contg. normal cells or tumour cells having cell-associated antigens capable of eliciting an immunological response and having contained DNA or RNA covalently bonded to psoralen or a psoralen deriv. in the presence of a long wavelength uv light (LW UV). The virus is suitably Herpes Simplex virus or influenza virus. The cells may be rabbit kidney cells or Madin Darby canine kidney cells.

ADVANTAGE - Use of psoralen (deriv.) and LWUV enables infectivity of cells to be eliminated rapidly using low amts. of radiant energy. All cells initially present can be recovered and native antigenicity is preserved. @(8pp

Abstract (EP): 8609 EP 66886

Immunologically reactive particles comprising inactivated, virus-infected cells having cell-associated antigens capable of eliciting an immunological response and having DNA or RNA which contains covalently bonded psoralen or a psoralen derivative whereby said inactivated target cells are non-infective, said virus-infected cells are normal or ***tumoral*** cells which have been infected by a virus. @(13pp)@

Derwent Class: B04; D16; S03; S05; R16;

File 155:MEDLINE(R) 1966-1994/Nov W4
(c) format only 1994 Dialog Info.Svcs.

Set Items Description
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?s kaplan(w)meir

1705 KAPLAN
18 MEIR
S1 2 KAPLAN(W)MEIR
?t s1/7/1-2

1/7/1
DIALOG(R)File 155:MEDLINE(R)
(c) format only 1994 Dialog Info.Svcs. All rts. reserv.

07694973 91213973
[Study of incidence and risk factors of nosocomial urinary tract infection in patients with indwelling urinary catheter in intensive care units]
Etude de l'incidence et des facteurs de risque de l'infection urinaire nosocomiale chez les malades sondes en reanimation polyvalente. Tasseau F; Chupin A; Pradier C; Villers D; Baron D; Nicolas F Service de reanimation medicale polyvalente, Centre hospitalier regional et universitaire de l'Hotel Dieu, Nantes.
Agressologie (FRANCE) 1990, 31 (8 Spec No) p503-4, ISSN 0002-1148 Journal Code: 31I
Languages: FRENCH Summary Languages: ENGLISH
Document type: JOURNAL ARTICLE English Abstract
A prospective study was carried out in a medical and surgical ICU to determine the incidence of nosocomial urinary tract infection (NUTI) and to identify the most important risk factors. Over a 6 month period, 180 patients were included. All had an indwelling catheter. Six risk factors were studied: age, sex, illness (medical, surgical, trauma), hospital or extra-hospital origin, simplified acute physiology score and length of bladder catheterization. Forty three patients developed a NUTI. Length of bladder catheterization was the only significant different risk factor in infected and non-infected patients. ***Kaplan*** ***Meir*** analysis was used to determine time to development of NUTI. The risk rose from 19% for 5 day long catheterization to 50% for 14 day long catheterization.

1/7/2
DIALOG(R)File 155:MEDLINE(R)
(c) format only 1994 Dialog Info.Svcs. All rts. reserv.

07309361 90216361
Adjunctive therapy (whole body hyperthermia versus lonidamine) to total body irradiation for the treatment of favorable B-cell neoplasms: a report of two pilot clinical trials and laboratory investigations. Robins HI; Longo WL; Steeves RA; Cohen JD; Schmitt CL; Neville AJ; O'Keefe S; Lagoni R; Riggs C
University of Wisconsin Clinical Cancer Center, Madison 53792. Int J Radiat Oncol Biol Phys (UNITED STATES) Apr 1990, 18 (4) p909-20, ISSN 0360-3016 Journal Code: G97
Contract/Grant No.: R01 35361-04; RR 03186, RR, NCRR
Languages: ENGLISH
Document type: JOURNAL ARTICLE
Based on earlier clinical and preclinical investigations, we designed two different pilot trials for patients with nodular lymphoma or chronic lymphocytic leukemia. These studies evaluated the use of either 41.8 degrees C whole body hyperthermia (WBH), or the nonmyelosuppressive chemotherapeutic drug, lonidamine (LON), as an adjunct to total body irradiation (TBI) (12.5 cGy twice a week, every other week for a planned total dose of 150 cGy). Whole body hyperthermia was initiated approximately 10 min after

total body irradiation; lonidamine was administered orally (420 mg/m2) on a daily basis. Although entry to the studies was nonrandomized, the two patient populations were accrued during the same time frame and were comparable in terms of histology, stage of disease, performance status, and prior therapy. Of 8 patients entered on the TBI/WBH study, we observed 3 complete responses (CR), 4 partial responses (PR), and 1 improvement (i.e., a 48% decrease in tumor burden). Of 10 patients entered in the TBI/LON study, there was 1 CR and 4 PR. For the TBI/WBH study, myelosuppression was not treatment-limiting; there were no instances of infection or bleeding and platelet support was never required. The median survival time for the TBI/WBH study is 52.5 months based on ***Kaplan*** ***Meir*** estimates. Two patients remain in a CR. The median time to treatment failure (MTTF) is 9.4 months (90% confidence interval = 7-15.4 months). In the TBI/LON study, 50% of patients receiving TBI required treatment modification due to platelet-count depression during therapy, but there were no instances of infection or bleeding. Frequently observed LON-related toxicities included myalgias, testicular pain, photophobia and ototoxicity. For the TBI/LON study, median survival is 7.6 months; MTTF was 2.4 months. In analyzing the results of these pilot studies, our subjective clinical impressions lead to the hypothesis that WBH protected against TBI-induced thrombocytopenia during therapy, whereas LON had no effect on TBI-induced myelosuppression. This speculation was tested and confirmed in a series of in vitro and in vivo experiments. ?b 155,5,73

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     06oct94 09:52:46 User219549 Session B68.2
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  File 155:MEDLINE(R)  1966-1994/Nov W4
       (c) format only 1994 Dialog Info.Svcs.
  File  5:BIOSIS PREVIEWS(R)  1969-1994/OCT W4
       (c) 1994 BIOSIS
  File 73:EMBASE  1974-1994/ISS 39
       (c) 1994 Elsevier Science B.V.
*File 73: Truncate EMTREE codes(e.g. DC=C1.120?) for complete retrieval.  See HELP NEWS 73 for
explode feature.

     Set  Items  Description
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?s kaplan(w)meir

          4401  KAPLAN
            62  MEIR
     S1     11  KAPLAN(W)MEIR
?rd

...completed examining records
     S2      9  RD (unique items)
?t s2/7/1-9


 2/7/1     (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 1994 Dialog Info.Svcs. All rts. reserv.

07694973   91213973
  [Study of incidence and risk factors of nosocomial urinary tract infection in patients with indwelling urinary catheter in intensive care units]
  Etude de l'incidence et des facteurs de risque de l'infection urinaire nosocomiale chez les malades sondes en reanimation polyvalente.  Tasseau F; Chupin A; Pradier C; Villers D; Baron D; Nicolas F  Service de reanimation medicale polyvalente, Centre hospitalier regional et universitaire de l'Hotel Dieu, Nantes.
  Agressologie (FRANCE)  1990,  31 (8 Spec No) p503-4,  ISSN 0002-1148 Journal Code: 31I
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Languages: FRENCH Summary Languages: ENGLISH
Document type: JOURNAL ARTICLE English Abstract
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2/7/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07309361 90216361
Adjunctive therapy (whole body hyperthermia versus lonidamine) to total body irradiation for the treatment of favorable B-cell neoplasms: a report of two pilot clinical trials and laboratory investigations. Robins HI; Longo WL; Steeves RA; Cohen JD; Schmitt CL; Neville AJ; O'Keefe S; Lagoni R; Riggs C
University of Wisconsin Clinical Cancer Center, Madison 53792. Int J Radiat Oncol Biol Phys (UNITED STATES) Apr 1990, 18 (4) p909-20, ISSN 0360-3016 Journal Code: G97
Contract/Grant No.: R01 35361-04; RR 03186, RR, NCRR
Languages: ENGLISH
Document type: JOURNAL ARTICLE
Based on earlier clinical and preclinical investigations, we designed two different pilot trials for patients with nodular lymphoma or chronic lymphocytic leukemia. These studies evaluated the use of either 41.8 degrees C whole body hyperthermia (WBH), or the nonmyelosuppressive chemotherapeutic drug, lonidamine (LON), as an adjunct to total body irradiation (TBI) (12.5 cGy twice a week, every other week for a planned total dose of 150 cGy). Whole body hyperthermia was initiated approximately 10 min after total body irradiation; lonidamine was administered orally (420 mg/m2) on a daily basis. Although entry to the studies was nonrandomized, the two patient populations were accrued during the same time frame and were comparable in terms of histology, stage of disease, performance status, and prior therapy. Of 8 patients entered on the TBI/WBH study, we observed 3 complete responses (CR), 4 partial responses (PR), and 1 improvement (i.e., a 48% decrease in tumor burden). Of 10 patients entered in the TBI/LON study, there was 1 CR and 4 PR. For the TBI/WBH study, myelosuppression was not treatment-limiting; there were no instances of infection or bleeding and platelet support was never required. The median survival time for the TBI/WBH study is 52.5 months based on ***Kaplan*** ***Meir*** estimates. Two patients remain in a CR. The median time to treatment failure (MTTF) is 9.4 months (90% confidence interval = 7-15.4 months). In the TBI/LON study, 50% of patients receiving TBI required treatment modification due to platelet-count depression during therapy, but there were no instances of infection or bleeding. Frequently observed LON-related toxicities included myalgias, testicular pain, photophobia and ototoxicity. For the TBI/LON study, median survival is 7.6 months; MTTF was 2.4 months. In analyzing the results of these pilot studies, our subjective clinical impressions lead to the hypothesis that WBH protected against TBI-induced thrombocytopenia during therapy, whereas LON had no effect on TBI-induced myelosuppression. This speculation was tested and confirmed in a series of in vitro and in vivo experiments.

2/7/3 (Item 1 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)

9581745 BIOSIS Number: 94086745
NATURAL HISTORY OF SMALL UNTREATED HEPATOCELLULAR CARCINOMA IN CIRRHOSIS A MULTIVARIATE ANALYSIS OF PROGNOSTIC FACTORS OF TUMOR GROWTH RATE AND PATIENT SURVIVAL

BARBARA L; BENZI G; GAIANI S; FUSCONI F; ZIRONI G; SIRINGO S; RIGAMONTI A ; BARBARA C; GRIGIONI W; ET AL

INQ.: LUIGI BOLONDI, IST. CLIN. MED. GASTROENTEROL., POLICLINICO S. ORSOLA, VIA MASSARENTI 9, 40138 BOLOGNA, ITALY.

HEPATOLOGY 16 (1). 1992. 132-137. CODEN: HPTLD

Full Journal Title: HEPATOLOGY (Baltimore)

Language: ENGLISH

We analyzed the growth pattern of tumor masses and the survival of 39 asymptomatic Italian patients with a total of 59 small (.ltoreq. 5 cm in diameter) hepatocellular carcinomas arising from cirrhosis. The total length of the observation period ranged from 90 to 962 days, with an average of 364 .+-. 229 (mean .+-. S.D.). Doublng time ranged from 27.2 to 605.6 days (mean .+-. S.D., 204.2 .+-. 135; median = 171.6 days). Three different growth patterns were recognized: (a) tumors with no or very slow initial growth pattern (doubling time > 200 days), 10 cases (37%); (b) tumors with declining growth rate over time, 9 cases (33.4%); and (c) tumors with almost constant growth rate, 8 cases (29.6%). Using the stepwise discriminant analysis, we found a score based on albumin, alcohol intake, number of nodules, echo pattern and histological type that allowed a correct prediction of short doubling time (.ltoreq. 150 days) in 55.6%, medium double time (151 to 300 days) in 60% and long doubling time (> 300 days) in 100% of cases. The estimated survival rate of the 39 patients, calculated by the Kaplan-Meier method was 81% at 1 yr, 55.7% at 2 yr and 21% at 3 yr. Stepwise discriminant analysis showed that a score based on sex, HBsAg status, alcohol consumption, ascites, .gamma.-glutamyltranspepti dase, prothrombin time, Child-Pugh class and all the sonographical parameters could predict 2-yr survival in 100% of cases. We conclude that great variability of growth patterns exist among and within small hepatocellular carcinomas. Prediction of subsequent growth rate is unreliable in most cases. The sonographical characteristics, together with the histological features, can, however, help in identifying cases with long doubling times (> 300 days). The discrimnant analysis on survival of cirrhotic patients with small hepatocellular carcinomas demonstrates that the underlying liver disease plays a key role in the long-term survival probability.

2/7/4 (Item 2 from file: 5)

DIALOG(R)File 5:BIOSIS PREVIEWS(R)

8130652 BIOSIS Number: 91051652

COURSE OF HIV-I INFECTION IN A COHORT OF HOMOSEXUAL AND BISEXUAL MEN AN 11 YEAR FOLLOW-UP STUDY

RUTHERFORD G W; LIFSON A R; HESSOL N A; DARROW W W; O'MALLEY P M; BUCHBINDER S P; BARNHART J L; BODECKER T W; CANNON L

INFECT. DISEASE BRANCH, CALIFORNIA DEP. HEALTH SERVICES, 2151 BERKELEY WAY, ROOM 708, BERKELEY, CALIFORNIA 94704, UNITED STATES.

BR MED J 301 (6762). 1990. 1183-1188. CODEN: BMJOA

Full Journal Title: British Medical Journal

Language: ENGLISH

Objective: To characterise the natural history of sexually transmitted HIV-I infection in homosexual and bisexual men. Design: Cohort study. Setting: San Francisco municipal sexually transmitted disease clinic. Patients: Cohort included 6705 homosexual and bisexual men originally recruited from 1978 to 1980 for studies of sexually transmitted hepatitis B. This analysis is of 489 cohort members who were either HIV-I seropositive on entry into the cohort (n=312) or seroconverted during the study period and had .ltoreq. 24 months between the dates of their last seronegative and first seropositive specimens (n=177). A subset of 442 of these men was examined in 1988 or 1989 or had been reported to have developed AIDS. Main outcome measures: Development of clinical signs and symptoms of HIV-I infection, including AIDS, AIDS related complex, asymptomatic generalised lymphadenopathy, and no signs or symptoms of infection. Measurements and main results: Of the 422 men examined in 1988 or 1989 or reported as having AIDS, 341 had been infected from 1977 to 1980; 49% (167) of these men had died of AIDS, 10% (34) were alive with AIDS, 19% (65) had AIDS related complex, 3% (10) had asymptomatic

generalised lymphadenopathy, and 19% (34) had no clinical signs or symptoms of HIV-I infection. Cumulative risk of AIDS by duration of HIV-I infection was analysed for all 489 men by the ***Kaplan***-***Meir*** method. Of these 489 men, 226 (46%) had been diagnosed as having AIDS. We estimated that 13% of cohort members will have developed AIDS within five years of seroconversion, 51% within 10 years, and 54% within 11.1 years. Conclusion: Our analysis confirming the importance of duration of infection to clinical state and the high risk of AIDS after infection underscores the importance of continuing efforts both to prevent transmission of HIV-I and to develop further treatments to slow or stall the progression of HIV-I infection to AIDS.

2/7/5 (Item 3 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)

7160085 BIOSIS Number: 88082830
RISK FACTORS AND PROGNOSIS IN PRIMARY BILIARY CIRRHOSIS
GOUDIE G M; BURT A D; MACFARLANE G J; BOYLE P; GILLIS C R; MACSWEEN N M; WATKINSON G
UNIV. DEP. PATHOL., WESTERN INFIRMARY, GLASGOW G11 6NT, SCOTLAND. AM J GASTROENTEROL 84 (7). 1989. 713-716. CODEN: AJGAA Full Journal Title: American Journal of Gastroenterology Language: ENGLISH
The relationship between survival and 25 clinical and histologic variables was studied in 195 patients (171 women, 24 men) who satisfied stringent criteria for the diagnosis of primary biliary cirrhosis. The mean duration of follow-up was 6 yr (range 0-17). One hundred and sixteen patients died, 84% as the result of liver disease and 16% from nonhepatic causes. Using the ***Kaplan***-***Meir*** estimate, we calculated the mortality from liver disease to be 40% after 5 yr and 60% after 10 yr. Ascites, serum bilirubin level, variceal hemorrhage, and age were identified as independent clinical risk factors, and extent of hepatic fibrosis, bilirubinostasis, and Mallory's hyalin were identified as independent histologic risk factors correlating with reduced survival.

2/7/6 (Item 4 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)

6497606 BIOSIS Number: 85098127
ACUTE MYOCARDIAL INFARCTION ASSOCIATED WITH SINGLE VESSEL CORONARY ARTERY DISEASE AN ANALYSIS OF CLINICAL OUTCOME AND THE PROGNOSTIC IMPORTANCE OF VESSEL PATENCY AND RESIDUAL ISCHEMIC MYOCARDIUM
WILSON W W; GIBSON R S; NYGAARD T W; CRADDOCK G B JR; WATSON D D; CRAMPTON R S; BELLER G A
DIV. CARDIOL., BOX 158, UNIV. VA. MED. CENT., CHARLOTTESVILLE, VA. 22908. J AM COLL CARDIOL 11 (2). 1988. 223-234. CODEN: JACCD Full Journal Title: Journal of the American College of Cardiology Language: ENGLISH
The long-term outcome and the significance of residual ischemic myocardium, as assessed by predischarge exercise thallium scintigraphy and vessel patency, were studied in 97 patients with single vessel coronary artery disease by angiography 12 .+-. 4 days after uncomplicated myocardial infarction. During a mean follow-up period of 39 .+-. 17 months, no patients died, 6 (6%) had a recurrent nonfatal infarction and 25 (26%) experienced rapidly progressive angina requiring hospitalization. Although neither exercise-induced angina nor ST segment depression was predictive of a recurrent cardiac event, the mean number of infarct zone scan segments showing thallium redistribution (1.0 .+-. 1.0 versus 0.5 .+-. 0.8, p = 0.01) and the percent of patients with infarct zone redistribution (61 versus 39%, p = 0.05) were greater in those patients who experienced a late ischemic event. ***Kaplan***-***Meir*** analysis demonstrated a lower event-free survival rate in patients with redistribution (n = 45) than in those without redistribution (n = 52) (p = 0.019). Although no patient received immediate thrombolytic therapy, the infarct-related vessel

was angiographically patent in 40 patients (41%). Vessel patency did not influence event-free survival, although a patent vessel, as compared with an occluded vessel, was associated with a greater prevalence of non-Q wave infarction (58 versus 21%, p < 0.001), fewer persistent infarct zone thallium defects (1.2 .+-. 1.1 versus 2.0 .+-. 1.2, p = 0.001), more reversible infarct zone thallium defects (1.0 .+-. 1.0 versus 0.5 .+-. 0.9, p = 0.02) and a trend toward a higher left ventricular ejection fraction (53 .+-. 10% versus 49 .+-. 12%, p = 0.07). In summary, uncomplicated myocardial infarction in patients with single vessel coronary artery disease is associated with a very low incidence of subsequent death and reinfarction. The presence of infarct zone thallium redistribution, compared with its absence, is predictive of a higher cardiac event rate. These data should be considered when recommending prophylactic percutaneous transluminal angioplasty after uncomplicated myocardial infarction in asymptomatic patients with single vessel coronary disease. On the basis of these results, future randomized trials designed to evaluate the therapeutic efficacy of revascularization in asymptomatic postinfarction patients with single vessel disease should limit enrollment to those patients with residual ischemia located within the infarct zone.

2/7/7 (Item 5 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)

5479715 BIOSIS Number: 32002022
THE TIME FROM INFECTION WITH HUMAN IMMUNODEFICIENCY VIRUS TO THE ONSET OF ACQUIRED IMMUNODEFICIENCY SYNDROME
TAYLOR J M G; SCHWARTZ K; DETELS R
DIV. BIOSTATISTICS, SCH. PUBLIC HEALTH, UCLA, LOS ANGELES, CALIF. 90024. J INFECT DIS 154 (4). 1986. 694-697. CODEN: JIDIA
Full Journal Title: Journal of Infectious Diseases
Language: ENGLISH

2/7/8 (Item 6 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)

3240280 BIOSIS Number: 21032683
STATISTICAL TECHNIQUE FOR EVALUATING LONGEVITY IN SHEEP
OLTHOFF J C; BOYLAN W J; HINKLEY D V
UNIV. MINN., ST. PAUL.
72ND ANNUAL MEETING OF THE AMERICAN SOCIETY OF ANIMAL SCIENCE, ITHACA, N.Y., USA, JULY 27-30, 1980. J ANIM SCI 51 (SUPPL. 1). 1980 (RECD. 1981). 127. CODEN: JANSA
Language: ENGLISH

2/7/9 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

9310891 EMBASE No: 94265701
Early- and late-onset pelvic inflammatory disease among women with cervical Chlamydia trachomatis infection at the time of induced abortion - A follow-up study
Sorensen J.L.; Thranov I.; Hoff G.; Dirach J.
PO Box 1975, DK-1023 Copenhagen K Denmark
INFECTION (Germany) , 1994, 22/4 (242-246) CODEN: IFTNA ISSN: 0300-8126
LANGUAGES: English SUMMARY LANGUAGES: English; German
After termination of a double-blind, randomized study on erythromycin in the prevention of post-abortion infection, 34 women (14 treated with erythromycin, 20 not treated with erythromycin) harbouring

Chlamydia trachomatis were followed up with 6 weeks and again 2 to 24 months after the abortion in order to detect an early- and late-onset pelvic inflammatory disease (PID). For statistical analysis survival analysis by ***Kaplan***-***Meir*** estimates and Mantel-Cox test were carried out. Untreated women with C. trachomatis infection at the time of abortion had a cumulative risk of 72% of developing early and/or late PID, if observed for 24 months. This cumulative risk was significantly reduced to 8% if the C. trachomatis infection was treated at the time of the abortion. Screening for and treatment of C. trachomatis is warranted, especially in women less than or equal to 25 years old, to avoid early and late-onset PID after induced first trimester abortion.

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?b 155

  File 155:MEDLINE(R) 1966-1994/Nov W4
    (c) format only 1994 Dialog Info.Svcs.

    Set  Items  Description
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?s dinitrophenyl or dnp

         1576  DINITROPHENYL
         2760  DNP
    S1   3716  DINITROPHENYL OR DNP
?e dinitrofluorobenzene


Ref   Items   RT  Index-term
E1        2       DINITROFLUOROBENZE
E2        1       DINITROFLUOROBENZEND
E3      863    6 *DINITROFLUOROBENZENE
E4       35       DINITROFLUOROBENZENE --ADMINISTRATION AND DOSA E5       10
DINITROFLUOROBENZENE --ADVERSE EFFECTS --AE E6       57       DINITROFLUOROBENZENE
--ANALOGS AND DERIVATIVES E7        4       DINITROFLUOROBENZENE --ANALYSIS --AN
E8        3       DINITROFLUOROBENZENE --ANTAGONISTS AND INHIBIT E9        5
DINITROFLUOROBENZENE --BLOOD --BL
E10       1       DINITROFLUOROBENZENE --CHEMICAL SYNTHESIS --CS E11       3
DINITROFLUOROBENZENE --CHEMISTRY --CH
E12      23       DINITROFLUOROBENZENE --DIAGNOSTIC USE --DU
       Enter P or PAGE for more
?s e3 and (cancer or tumor)

          863  DINITROFLUOROBENZENE
       188855  CANCER
       233518  TUMOR
    S2     49  "DINITROFLUOROBENZENE" AND (CANCER OR TUMOR) ?rd


  > > > Duplicate detection halted.

?s  s2 an s1

> > > Term "AN" in invalid position
?s  s2 and s1

          49  S2
        3716  S1
    S3     1  S2 AND S1
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?t s3/6/1

3/6/1
08902446 94217446
The effect of anti-***tumor*** necrosis factor (TNF)-alpha monoclonal antibody on allergic cutaneous late phase reaction in mice. ?t s3/7/1

3/7/1
DIALOG(R)File 155:MEDLINE(R)

08902446 94217446
The effect of anti-***tumor*** necrosis factor (TNF)-alpha monoclonal antibody on allergic cutaneous late phase reaction in mice. Sakurai T; Inagaki N; Nagai H
Department of Pharmacology, Gifu Pharmaceutical University, Japan. Life Sci (ENGLAND) 1994, 54 (17) pPL291-5, ISSN 0024-3205 Journal Code: L62
Languages: ENGLISH
Document type: JOURNAL ARTICLE
Biphasic cutaneous reaction with peak response at 1 (early phase) and 24 to 48 hour (late phase) was elicited by epicutaneous challenge with antigen in actively and passively sensitized mice. Mice were actively immunized with dinitrophenylated (***DNP***) ascaris antigen and challenged with ***dinitrofluorobenzene*** (DNFB). Passively sensitization was carried out by the injection of monoclonal anti-***DNP***-IgE antibody into mice and challenge was elicited with DNFB. Prednisolone at doses of 3 to 10 mg/kg clearly inhibited both early and late phase reactions in either sensitized mice. Monoclonal anti-***tumor*** necrosis factor (TNF)-alpha antibody inhibited the late phase cutaneous reaction in actively sensitized mice. Anti-interleukin-5 (IL-5) monoclonal antibody has no effect on both phase reactions in either actively and passively sensitized animals. These results indicate the possible participation of TNF-alpha in allergic cutaneous late phase reaction in actively sensitized mice. ?s s1 and (dinitrofluorobenzene or dnfb)

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        3716 S1
         863 DINITROFLUOROBENZENE
         339 DNFB
    S4   137 S1 AND (DINITROFLUOROBENZENE OR DNFB)
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?s s4 and (desensitiz? or sensitiz?)

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         137 S4
       11623 DESENSITIZ?
       26829 SENSITIZ?
    S5    44 S4 AND (DESENSITIZ? OR SENSITIZ?)
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?t s5/6/1-44

5/6/1
08?t s5/7/13,18,19,21,31,36,42,43

5/7/13
DIALOG(R)File 155:MEDLINE(R)

06028572 87002572

Desensitization of contact allergy to ***DNFB*** in mice. III. Characteristics of immediate ***desensitization*** induced by haptenated spleen cells.

Mekori YA; Claman HN

Cell Immunol (UNITED STATES) Apr 1 1986, 98 (2) p279-88, ISSN 0008-8749 Journal Code: CQ9

Contract/Grant No.: AI-12685

Languages: ENGLISH

Document type: JOURNAL ARTICLE

The immediate effects and mechanisms of ***desensitization*** of contact sensitivity to ***dinitrofluorobenzene*** (***DNFB***) were investigated. Intravenous injection of dinitrophenol (***DNP***)-labeled syngeneic spleen cells caused significant antigen-specific inhibition (greater than 40%) of the contact response within 24 hr in mice that had been ***sensitized*** 2 weeks prior to ***desensitization***. With low concentrations of the hapten used for labeling, allogeneic spleen cells were found to be more efficient than syngeneic ones in inducing the down-regulation of the contact response. The most efficient ***desensitization*** was produced by ***DNP*** -cells that differed from the recipient at the MLS locus. Haptenated spleen cells induced the production of suppressor mechanisms, as spleen cells from animals ***desensitized*** with ***DNP***-cells were able to down-regulate recipients in adoptive transfer and could block the passive transfer of contact sensitivity. Procedures that interfere with the development of suppressor cells, e.g., cyclophosphamide and adult thymectomy, interfered with ***desensitization*** by ***DNP***-cells. These results are in contrast to ***desensitization*** with soluble dinitrobenzenesulfonic acid (DNBS), where suppressor mechanisms have not been shown. The mechanisms for ***desensitization*** depend upon the form of the ***desensitizing*** antigen.

5/7/18

DIALOG(R)File 155:MEDLINE(R)

05487877 85103877

Hapten-specific prophylaxis of murine contact sensitivity by ***DNP*** -amino acid derivatives.

Katayama I; Nishioka K

Int Arch Allergy Appl Immunol (SWITZERLAND) 1985, 76 (2) p101-6, ISSN 0020-5915 Journal Code: GP9

Languages: ENGLISH

Document type: JOURNAL ARTICLE

In mice, the elicitation of ***DNFB*** contact skin reaction was suppressed by the intravenous injection of relatively small amounts of ***DNP*** -amino acid conjugates when given at the same time of challenge test. Maximal suppression achieved was around 70% when optimal doses of ***DNP*** -amino acid were administered. The suppressive activity of 4 different amino acids, i.e., ***DNP***-L-lysine, ***DNP***-S-cysteine, ***DNP***-alanine, and ***DNP*** -glycine, was nearly the same, respectively. This inhibitory effect was hapten-specific, and it failed to suppress the strong skin reaction elicited in cyclophosphamide-pretreated mice. Using the lymphocyte culture system, low doses of ***DNP***-amino acid also suppressed the antigen-specific proliferation of ***sensitized*** lymph node cells (T prlf). Preincubation of ***sensitized*** cells with ***DNP*** -amino acid eliminated the transfer activity of T-DH cells as demonstrated previously. The mixture of lymph node cells and spleen cells from ***DNP*** -L-lysine-treated mice in vivo also suppressed the elicitation of skin reaction when transferred to recipient mice ***sensitized*** 5 days previously. Transfer studies demonstrated that this suppression was mediated by Lyt 1.2 (+), Thy 1.2 positive cells in vivo.

5/7/19

DIALOG(R)File 155:MEDLINE(R)

05408973 85024973

Desensitization of contract allergy to 2,4-dinitro-1-fluorobenzene in mice. II. Characteristics of "immediate" ***desensitization***. Mekori YA; Claman HN
Cell Immunol (UNITED STATES) Nov 1984, 89 (1) p84-94, ISSN 0008-8749 Journal Code: CQ9
Contract/Grant No.: 5 RO1 AI-12685
Languages: ENGLISH
Document type: JOURNAL ARTICLE
The immediate effects and mechanisms of ***desensitization*** of contact sensitivity to 2,4-dinitro-1-fluorobenzene (***DNFB***) were investigated. Mice were ***sensitized*** with ***DNFB***, ***desensitized*** with antigen 2 weeks later, and challenged 1 day after ***desensitization*** . Significant inhibition (approximately 50%) of contact sensitivity was observed after iv injections of large doses of dinitrobenzene sulfonic acid (DNBS) or dinitropenol (***DNP***)-labeled spleen cells. Haptenated red blood cells (RBC) did not induce any significant immediate ***desensitization*** but produced significant inhibition of an anamnestic response 2 weeks later. The immediate ***desensitization*** induced by DNBS was antigen nonspecific. Although the contact sensitivity response itself could be inhibited by afferent- or efferent-acting suppressor cells, such cells were not demonstrated in ***desensitized*** animals. DNBS appears to ***desensitize*** by inactivating effector cells for contact sensitivity, although it appears that suppressor mechanisms could be activated by other physiochemical forms of the ***desensitizing*** antigen.

5/7/21
DIALOG(R)File 155:MEDLINE(R)

05143533 84067533
Elicitation of delayed type hypersensitivity in chicks after in ovo ***sensitization*** with different molecular forms of the same hapten. Moriya O; Ichikawa Y
Microbiol Immunol (JAPAN) 1983, 27 (9) p779-85, ISSN 0385-5600 Journal Code: MX7
Languages: ENGLISH
Document type: JOURNAL ARTICLE
Lymphocyte ***sensitization*** , which participates in delayed type hypersensitivity (DTH) in chick embryos, was studied. The in ovo injection of dinitrophenylated keyhole limpet hemocyanin (***DNP***-KLH) or ***DNP*** -dextran (***DNP***-D) led to delayed onset of the hapten-specific reaction as shown by allergic contact dermatitis (ACD) after hatching. The extent of the ACD response was not directly dependent on the antigen dosage or the number of injections given for ***sensitizing***. The magnitude of the ACD response was higher in chicks ***sensitized*** with ***DNP***-D than in those given ***DNP***-KLH. These findings suggest the presence of embryonic lymphocytes which can be ***sensitized*** by in ovo antigenic stimulation at the later stage of embryogenesis and may make possible the differentiation of functional lymphocytes. Antigen stimulation with higher doses may be inadequate for the in ovo ***sensitization*** of embryonic lymphocytes. The ACD response elicited by ***DNFB*** in chicks primed with either ***DNP***-KLH or ***DNP*** -D was thought to be T-cell dependent, from the kinetics of the ACD peak within a period of 24 to 48 hr. Furthermore the conditions for in ovo ***sensitization*** of embryonic lymphocytes by ***DNP***-D seem to be different from those for ***sensitization*** by ***DNP***-KLH.

5/7/31
DIALOG(R)File 155:MEDLINE(R)

04092201 80203201
Hapten-cell conjugates in DNCB contact sensitivity. In vitro stimulation with ***DNP***-conjugates optimally inducing contact sensitivity in vivo. von Blomberg M; Scheper RJ
Immunology (ENGLAND) Feb 1980, 39 (2) p291-9, ISSN 0019-2805 Journal Code: GH7

Languages: ENGLISH
Document type: JOURNAL ARTICLE
Until now in vitro stimulation tests have failed to detect DNCB contact sensitivity in guinea-pigs ***sensitized*** epicutaneously without the use of adjuvants. In this study ***DNP***-conjugates optimally inducing contact sensitivity in vivo were tested for their capacity to detect DNCB contact sensitivity in vitro in a lymphocyte transformation assay. In vivo contact sensitivity measurements in guinea-pigs which had been immunized with different hapten-cell conjugates, showed that (1) living cells should be used for conjugation with the allergen, (2) macrophages are optimal carriers and (3) syngeneity is not required. Therefore, ***DNFB***-coated peritoneal macrophages (viable when conjugated) were used as an antigen for in vitro stimulation. Using this conjugate, a highly reproducible antigen-specific increase in DNA synthesis could be obtained in lymph node lymphocytes from guinea-pigs that had been ***sensitized*** to DNCB by epicutaneous application of the allergen without the use of adjuvants.

5/7/36
DIALOG(R)File 155:MEDLINE(R)

03411031 78045031
Anti-***DNP*** antibody response after the topical application of ***DNFB*** in mice.
Takahashi C; Nishikawa S; Katsura Y; Izumi T
Immunology (ENGLAND) Oct 1977, 33 (4) p589-96, ISSN 0019-2805 Journal Code: GH7
Languages: ENGLISH
Document type: JOURNAL ARTICLE
A single painting or daily paintings for 5 days with ***dinitrofluorobenzene*** (***DNFB***) on the abdominal skin of mice induced both contact sensitivity, detectable by ear swelling, and, hapten-reactive helper T cells, detectable by the augmented anti-bovine serum albumin (BSA) antibody response on challenge with ***dinitrophenyl*** -BSA. Contact sensitivity was induced within 7 days and helper activity within 14 days after the ***sensitization***. Anti-hapten antibody response in the spleen or regional lymph nodes of such mice, however, was negligibly small during the 15 days after a single painting. Failure to respond with anti-hapten antibody production of mice given only a single painting was shown to be due to the shortage of B cells reactive to the hapten. Daily paintings for 5 days did not necessarily result in the augmented antibody response. By contrast, a strong anti-hapten antibody response was observed in mice receiving two paintings at an interval of 10 days. In these mice, hapten-specific B memory cells as well as hapten-reactive T cells were detected. Thus, the anti-hapten antibody response after the topical applciation of simple chemicals may depend upon the priming of B cells, and the response must be mediated by the cooperation of T and B cells both reactive to the antigen.

5/7/42
DIALOG(R)File 155:MEDLINE(R)

03031064 76212064
Hapten-specific responses to contact ***sensitizers*** . Use of fluorodinitrobenzene to elicit migration inhibition and macrophage agglutination factors from lymph node cells of contact-sensitive guinea-pigs.
Godfrey HP
Immunology (ENGLAND) May 1976, 30 (5) p685-94, ISSN 0019-2805 Journal Code: GH7
Languages: ENGLISH
Document type: JOURNAL ARTICLE
Hapten-specific sensitivity of guinea-pigs ***sensitized*** to ***dinitrophenyl*** (***DNP***) contactants and to ***DNP*** -protein conjugates was investigated by skin test and by antigen-induced elaboration of migration inhibition (MIF) and macrophage agglutination factors (MAF) From lymph node cells. The delayed contact reaction was highly specific for low doses of contactant and markedly less so for

conjugates; lymph node cells elaborted both lymphokines in response to brief exposures to ***dinitrofluorobenzene*** (***DNFB***) or prolonged exposures to ***DNP*** conjugates. Elicitation of MAF by ***DNFB*** or ***DNP*** conjugate was inhibited in the presence of ***DNP*** glycine; the activity of MAF induced by ***DNP*** conjugate (but not that induced by ***DNFB***) was inhibited in the presence of ***DNP***-glycine as well. These results suggest that contact sensitivity to ***DNP*** conjugates reflect two different types of hapten-specific cellular sensitivity mediated by populations of cells with different antigen receptors and possibly, functionally different lymphokines.

5/7/43
DIALOG(R)File 155:MEDLINE(R)

02963628 76144628
Tolerance and contact sensitivity to ***DNFB*** in mice. V. Induction of tolerance with ***DNP*** compounds and with free and membrane-associated ***DNFB***.
Claman HN
J Immunol (UNITED STATES) Mar 1976, 116 (3) p704-9, ISSN 0022-1767 Journal Code: IFB
Languages: ENGLISH
Document type: JOURNAL ARTICLE
Immunologic unresponsiveness (tolerance) was induced in a mouse model of contact ***sensitization*** to ***DNFB***. The ability to induce tolerance varied with the chemical reactivity of the tolerogen; ***DNFB*** was highly tolerogenic, DNBSO3 was moderately tolerogenic, and ***DNP***-lysine was not tolerogenic. Although ***DNFB*** is considered a highly reactive compound, tracer studies of injected ***DNFB*** showed that it was rapidly excreted. Further studies were therefore done with ***DNFB*** attached to mouse erythrocytes. Tolerance to ***DNFB*** -RBC was highly specific in vivo; mice tolerant to ***DNFB*** showed normal reactivity to TNCB (picryl chloride.) Cells of mice tolerant to ***DNFB***-RBC were also unresponsive to DNBSO3 in vitro. Tolerance to ***DNFB***, DNBSO3, and ***DNFB***-RBC all required time to develop, suggesting that an active process was involved. ?pause

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PLEASE ENTER A COMMAND OR BE LOGGED OFF IN 5 MINUTES
?b 155

File 155:MEDLINE(R) 1966-1994/Nov W4
(c) format only 1994 Dialog Info.Svcs.

Set	Items	Description
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	33237	SOLID
	303449	TUMOR?
	5355	SOLID(W)TUMOR?
S1	844	METASTA? AND SOLID(W)TUMOR?
?s s1 and (treat or treatment or therapy)		
Processing		
	844	S1
	17495	TREAT
	741697	TREATMENT

1196525 THERAPY
S2 486 S1 AND (TREAT OR TREATMENT OR THERAPY)
?s s2 and melanoma

486 S2
30510 MELANOMA
S3 75 S2 AND MELANOMA
?t s3/6/1-75

?t s3/7/11,20,31,43,70,72,74

3/7/11
DIALOG(R)File 155:MEDLINE(R)

08309418 93019418
Intravenous administration of recombinant human macrophage colony-stimulating factor to patients with ***metastatic*** cancer: a phase I study.
Sanda MG; Yang JC; Topalian SL; Groves ES; Childs A; Belfort R Jr; de Smet MD; Schwartzentruber DJ; White DE; Lotze MT; et al
Surgery Branch, National Cancer Institute, National Institutes of Health, Bethesda, MD.
J Clin Oncol (UNITED STATES) Oct 1992, 10 (10) p1643-9, ISSN 0732-183X Journal Code: JCO
Languages: ENGLISH
Document type: CLINICAL TRIAL; CLINICAL TRIAL, PHASE I; JOURNAL ARTICLE PURPOSE: Recombinant human macrophage colony-stimulating factor (M-CSF) has been shown to stimulate specifically macrophage lineage differentiation in vitro and to induce cells capable of antitumor activity alone or in combination with an antibody. The administration of M-CSF to mice has demonstrated antitumor therapeutic effects in vivo. Therefore, a phase I trial of M-CSF administration to patients with ***metastatic*** cancer was undertaken. PATIENTS AND METHODS: M-CSF was given by intermittent intravenous bolus infusion every 8 hours for 7 days; the ***treatment*** cycle was repeated once after a week of rest. Cohorts of three patients underwent dose escalation from 10 to 100,000 micrograms/m2/d; 23 patients received 27 courses of M-CSF administration. All patients had ***metastatic*** ***solid*** ***tumors*** refractory to conventional ***therapy***, including renal cell carcinoma (RCC) (nine), ***melanoma*** (seven), and colorectal carcinoma (seven). RESULTS: ***Treatment*** -related toxicity was minimal; five patients developed transient signs of ocular or periorbital inflammation, with iridocyclitis as the most severe manifestation. At the highest doses, platelet counts decreased with ***therapy*** (but remained > 100,000/mm3) and the absolute monocyte count increased during the course of ***therapy***. Only at 30,000 and 100,000 micrograms/m2/d was ***treatment*** limited because of toxicity (iritis and malaise). Pharmacokinetic studies demonstrated up to a 1,000-fold increase in circulating serum M-CSF after bolus infusion; half-life varied from 1 to 6 hours. Complete regression of mediastinal adenopathy and multiple pulmonary ***metastases*** were observed in one patient with RCC. CONCLUSION: Recombinant M-CSF can be administered safely to patients with ***metastatic*** cancer at doses that demonstrate biologic activity.

3/7/20
DIALOG(R)File 155:MEDLINE(R)

07558026 91077026
New therapeutic strategies in the ***treatment*** of murine diseases induced by virus and ***solid*** ***tumors***: biology and implications for the potential ***treatment*** of human leukemia, AIDS, and

solid ***tumors***.

Shen RN; Lu L; Broxmeyer HE

Department of Radiation Oncology/Medicine, Walther Oncology Center, Indiana School of Medicine, Indianapolis.

Crit Rev Oncol Hematol (UNITED STATES) 1990, 10 (3) p253-65, ISSN 1040-8428 Journal Code: AGO

Languages: ENGLISH

Document type: JOURNAL ARTICLE; REVIEW; REVIEW, ACADEMIC Understanding the biology and ***treatment*** of various cancers (including leukemia) and immunodeficiency disorders is still an ongoing and experimental process. Animal models have been and continue to be important to this process. This review will focus in on work by ourselves and others that have used murine models assessing the effects in vivo of the Friend virus complex (FVC, composed of a spleen focus forming virus and a murine leukemia helper virus) and ***solid*** ***tumors*** with ***metastatic*** potential in order to evaluate new and innovative therapies. These therapies include radiation, hyperthermia, and newly recognized naturally occurring biomolecules termed cytokines. These cytokines include, but are not limited to, the interferons, the tumor necrosis factors, the interleukins, the hematopoietic colony stimulating factors, lactoferrin and E-type prostaglandins. For example, it has been found that lactoferrin, when administered early enough, prolongs the survival of mice injected, but not yet infected, with the FVC. Of even greater potential usefulness is that mice already infected with the FVC can be completely rescued from death by ***treatment*** with split low dosage (150 cGy) total body irradiation. Irradiation ***treatment*** was associated with restoration of the T helper to T suppressor cell ratio, natural killer cell activity and marrow proliferative responses to the mitogens PHA and con A which were compromised by the FVC. More recent studies in our laboratory have demonstrated the potential of the interleukins and colony stimulating factors to decrease the ***metastatic*** potential of the B16 ***melanoma*** and the Lewis Lung Carcinoma cell lines. The cytokines can act in greater than additive fashion and combinations of therapies are possible. This review is meant to increase the awareness of these investigative animal models and the new types of combination therapies that can then be used as the basis for future clinical trials evaluating therapeutic efficacy. (166 Refs.)

3/7/31

DIALOG(R)File 155:MEDLINE(R)

06875432 89177432

Interleukin-2 and lymphokine-activated killer cell ***therapy*** of ***solid*** ***tumors***: analysis of toxicity and management guidelines. Margolin KA; Rayner AA; Hawkins MJ; Atkins MB; Dutcher JP; Fisher RI; Weiss GR; Doroshow JH; Jaffe HS; Roper M; et al

Department of Medical Oncology, City of Hope National Medical Center, Duarte, CA 91010.

J Clin Oncol (UNITED STATES) Apr 1989, 7 (4) p486-98, ISSN 0732-183X Journal Code: JCO

Contract/Grant No.: N01 CM73702; N01 CM73703; N01 CM73704; + Languages: ENGLISH

Document type: CLINICAL TRIAL; JOURNAL ARTICLE; MULTICENTER STUDY The National Cancer Institute (NCI) Extramural IL2/LAK Working Group treated 93 patients with 114 cycles of high-dose intravenous (IV) interleukin-2 (IL-2) and lymphokine-activated killer (LAK) cells in three phase II trials. Thirty-six patients had ***metastatic*** ***melanoma***, 35 had ***metastatic*** renal cell cancer, and 22 had colorectal cancer. All patients had a Karnofsky performance status greater than or equal to 80% and normal laboratory tests and organ function, and had received no more than one prior form of immunotherapy or chemotherapy. Objective responders were eligible to receive up to two additional courses of ***therapy*** at 12-week intervals. The most frequent toxicities were a capillary leak syndrome resulting in marked extravascular fluid shifts, and hypotension requiring ***treatment*** with large volumes of IV fluids and vasopressor agents. Laboratory and clinical evidence of hepatic and renal dysfunction were virtually universal. Intensive care-level support was routinely provided and the toxicity observations confirmed the need for this level of care. The life-threatening toxicities were cardiac and pulmonary. Five of the 27 patients who experienced significant respiratory compromise required intubation and mechanical

ventilatory support. Twenty patients developed cardiac arrhythmias, the majority of which were supraventricular. There was a single episode of ventricular tachycardia requiring cardioversion. Four patients had transient cardiac ischemia, and an additional four had myocardial infarctions, one of which was fatal. With these exceptions, all toxicities were rapidly reversible. The occurrence of only a single ***therapy***-related death and a very low incidence of other irreversible or life-threatening events is comparable to the level of toxicities often observed in other phase II trials. Although the intensity of this regimen limits this approach to a subset of cancer patients with excellent performance status and adequate organ function, because of the frequency and apparent durability of complete responses, this ***treatment*** warrants further investigation.

3/7/43
DIALOG(R)File 155:MEDLINE(R)

06220264 87194264
The effect of kazusamycin on the growth of murine ***solid*** ***tumors*** and their spontaneous ***metastasis***.
Yoshida E; Nishimuta Y; Naito K; Watanabe Y; Tomisaka S; Okura A; Komiyama K; Umezawa I
J Antibiot (Tokyo) (JAPAN) Mar 1987, 40 (3) p391-3, ISSN 0021-8820 Journal Code: HCF
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